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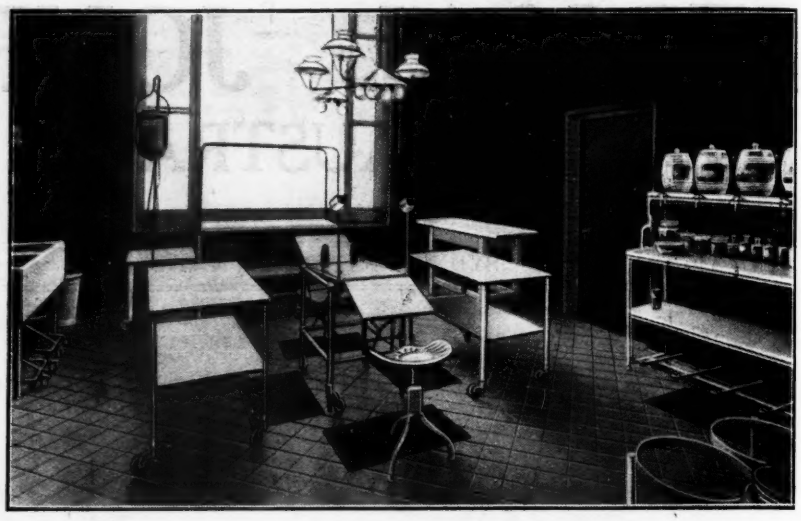

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Table of Contents

PAGE.

ORIGINAL ARTICLES—

"An Address," by G. W. BARBER, C.B., C.M.G., D.S.O., V.D., M.R.C.S., L.R.C.P. 539

"Dementia Præcox," by J. THEO. ANDERSON, L.R.C.P. & S.E. 540

"The Present Plague Outbreak in Sydney," by W. G. ARMSTRONG, M.B., CH.M., D.P.H. 546

"The Plague Epidemic," by E. W. FERGUSON, M.B., CH.M... 552

REVIEWS—

Medical Progress in China.. 554

LEADING ARTICLES—

Plague Anti-Serum 555

THE WEEK—

Hyperglycæmia and Diabetes 556

The Transmission of Measles to Guinea-Pigs .. 557

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—

Surgery 558

Gynæcology and Obstetrics 559

PAGE.

BRITISH MEDICAL ASSOCIATION NEWS—

Annual Meeting 560

Scientific 561

Nominations and Elections 564

Notices 564

OBITUARY—

Alfred Sutton 564

Archibald Lang McLean 565

CORRESPONDENCE—

The Cost of Medicines 565

Infantile Mortality 565

A Theory of Cancer 565

BOOKS RECEIVED 566

MEDICAL APPOINTMENTS 566

MEDICAL APPOINTMENTS VACANT, ETC. 566

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 566

DIARY FOR THE MONTH 566

EDITORIAL NOTICES 566

An Address.[1]

BY G. W. BARBER, C.B., C.M.G., D.S.O., V.D., M.R.C.S. (ENG.), L.R.C.P. (LOND.),

Retiring President of the Western Australian Branch of the British Medical Association.

THERE are two items that I would like to refer to at length. The first is the subject of the friendly societies' agreement and the second the *Hospital Bill.*

Friendly Society Practice.

In dealing with the Model Lodge Agreement, a great deal of work has been done, but I regret with not quite as satisfactory results as had been hoped for. To start with, your delegates had numerous meetings with the representatives of the Friendly Societies' Council, when the following rates were agreed upon: 24s. in the metropolitan area, 26s. at Kalgoorlie and Albany and 30s. in country districts. This agreement was made between the Western Australian Branch of the British Medical Association and the Friendly Societies' Council, whom the Branch believed represented all the friendly societies. It subsequently transpired that four of the smaller lodges were not represented and since then the Council has dealt directly with them. It was also understood that the Friendly Societies' Council had the full control of all the lodges under their Association, when on the other hand, it was presumed that our members would loyally adhere to any decision we arrived at.

To date eighty-two lodge agreements in accordance with the terms agreed upon have been duly signed by all parties. These, however, are principally in the metropolitan area. No agreements have come in from the Goldfields and it has been ascertained that the doctors and the friendly societies have made their own arrangements, although it is understood on the rate of pay which we agreed upon. The Council is still endeavouring to get all agreements on the prescribed form and hope they will be successful. I appeal to all members of the Branch to support the Council loyally in this matter, as it is entirely to their advantage. By doing so they will eliminate undercutting and it will be found that the majority of disputes can be amicably settled by the delegates from the respective councils, who meet when necessary for that purpose.

The Timber Mill Agreements were also the subject of recent consideration by your Council and it is hoped that the agreement now drafted will prove satisfactory and apply to all districts.

[1] Delivered at the Annual Meeting of the Western Australian Branch of the British Medical Association on March 26, 1922.

The Hospitals.

I must congratulate the Government on realizing that the medical arrangements for this State are unsatisfactory to the public and the medical profession.

A *Hospital Bill* was drafted, but referred to a Commission, as it met with universal disapproval, but this is only the first step and will, I hope, result in the Committee making recommendations which will result in a Bill acceptable to all. The arrangements for the cure and prevention of disease in this State are little better than they were twenty-five years ago, except in one respect—the Laboratory, which has proved a great boon. It is to be hoped that the Committee will ascertain the total sum required for increased accommodation, equipment which is lacking, and possible expansion and upkeep, not only of hospitals, but also of auxiliary institutions, such as the Children's Hospital, Home of Peace and Old Men's Home, all of which require investigation before deciding on the needs of the population. I have heard it stated that the Children's Hospital is larger than necessary and that the Home of Peace is far too small. The Old Men's Home, which the general public imagines is a home exclusively for old men who have been worsted in life's battle, also functions as a lunatic asylum, an infirmary for incurables, a convalescent home for the Perth Hospital, a sanatorium for persons suffering from miners' phthisis and a rest home for men temporarily out of work. It is obvious that very soon arrangements will have to be made to separate these various classes.

Further, I hope that the new Bill will include reference to transport of the sick and injured and some provision for medical inspection. A Medical Inspector of Hospitals has been appointed recently; but as his life's work is exclusively devoted to prevention of disease, I presume his activities will be limited to that aspect.

This very important matter of the *Hospital Bill* was given earnest consideration. Copies of the draft *Hospital Bill* were sent to all the members of the Branch, by direction of a general meeting, at great expense, but I regret to say not one reply was received nor a single suggestion made. Four of your members were appointed to attend the Parliamentary Select Committee on the matter and I think it was largely due to their evidence that the Bill was not passed. The fundamental principle of the Bill is not stated. The Government's duty is to provide a hospital for indigent patients (including accident and emergency cases), but the Bill does not state so. If the Bill is intended as a national measure for the treatment of sick members of the community, whether able to pay or not, it is not stated, nor is there any provision for the payment of medical officers? The Bill is most deficient, as it provides for no representation of the honorary medical staff on the Board of Management. The proposed method of taxation was unjust and would result, I am sure, in loss of efficiency. Apart from the honorary staff of the Perth Hospital and also the honorary staff at the Fremantle Hospital, it must be concluded that every district medical officer will be an honorary to his hospital where it exists and should be on the Board of Management for the reason that the district medical officer's pay, which includes the duty of attending members of the police force, aborigines, lunatics, etc., is the same whether there is a hospital in the district or not and the remuneration would be too small for it to include hospital treatment of indigent patients in the hospital. Any arrangements for the local doctor to take private patients into his hospital should be a private arrangement and not one to be included in any Act of Parliament.

The whole question of hospital systems demands thorough consideration. The public, who know nothing about the workings of a hospital, considers that all medical men are blessed with the ability of managing hospitals. It remains, however, for a few trained medical men and a few trained laymen to understand the administration of a hospital. I am an advocate of a system of hospitals and auxiliary institutions throughout the State for the treatment of indigent patients, graded and organized in equipment and staff as one organization and administered by a member of the profession. The tendency towards lay administration can only lead to trouble, decreased efficiency and increased expense.

I wish the Commission every success in their important undertaking and can assure them that the medical profession is keenly anxious for efficiency in the cure and prevention of disease in this State.

Emigration.

It may be desirable for the Council to consider the matter of emigration during the coming year. We have all noticed eulogistic references in the press to the extreme care taken in the selection of emigrants; but this is not correct. I, myself, during the past two years have seen a number of emigrants suffering from phthisis and in receipt of pensions from the Imperial Government on that account. I have seen others of indifferent physique and some suffering from disabilities precluding them from engaging in country pursuits. Many of these are, of course, suitable and desirable if proper arrangements are made for their reception beforehand; but they are not the type required in the country.

DEMENTIA PRÆCOX.[1]

BY J. THEO. ANDERSON, L.R.C.P. & S.E.,
Inspector-General of the Insane, Western Australia.

I KNOW of no term in psychiatry which is so faulty and so misleading as the term *dementia præcox* and certainly there is no term which has elicited such severe criticism. One may search the whole psychiatric literature in vain for a satisfactory definition. The word *dementia* means a permanent psychic disability due to neuronic degeneration. When that degeneration occurs, there can be no recovery, for, as far as our knowledge goes, destroyed nervous tissue cannot be re-established. The term *dementia,* even in this somewhat enlightened

[1] Read at the Annual Meeting of the Western Australian Branch of the British Medical Association on March 26, 1922.

period, is erroneously applied to anergia or stupor; it should be limited to the description of permanent loss of mental function.

The word *præcox* means youthful or premature. The obvious question to ask is: "When is dementia premature?" There is no dispute about senile dementia; but from the writings of some it can be concluded that a dementia occurring before the age of three score years and ten (the allotted span of life) should be regarded as premature, because some alienists have described cases of insanity occurring at the ripe age of sixty-three as forms of *dementia præcox*. If those views are correct, it follows as a *reductio ad absurdam* that there is practically only one form of insanity, *viz.*, *dementia præcox*, and that all other forms are subdivisions of this classification except, of course, senile dementia.

As regards the history of the term *dementia præcox*, Kahlbaum in 1863 described a form of mental disease occurring at puberty and rapidly terminating in dementia, which he called hebephrenia. In 1873 Clouston described similar cases as "insanity of adolescence" and in 1874 Kahlbaum described catatonia. These terms were not generally adopted; but in 1891 Pick introduced the much-debated term *dementia præcox* when he described cases including hebephrenia with maniacal symptoms varied by melancholia and rapid mental deterioration. Later Kraepelin showed the connexion between hebephrenia and the various forms of catatonia and embraced them under the title of *dementia præcox* and its varieties. In his "Lectures on Clinical Psychiatry," he devotes a lecture to each of the following subjects: *"Dementia præcox,"* "Catatonic Stupor," "Catatonic Excitement," "Paranoidal Forms of *dementia præcox*" and "The Final Stages of *dementia præcox*"; but makes it quite clear that he regards them not as separate entities, but as varieties of the same condition.

Clinically there does not appear to be any symptom complex that would correspond to or indicate any special process of mental deterioration; and pathologically there is no evidence of any special degenerative process in the brain tissue pathognomonic of *dementia præcox*. The term lacks description, for when an alienist is told that a patient is suffering from *dementia præcox*, it conveys no more to his mind than when a dermatologist is told that a patient is suffering from eczema. One writer has cleverly defined *dementia præcox* as a "vague and misleading term descriptive of a non-existent variety of insanity." He also states that "the medico-psychological dictionary of the future should have as an appendix a glossary of extinct terms—a kind of verbal cemetery in which *dementia præcox* with very many others might be decently interred with fitting monumental allusion to 'rest in peace,' without hope of resurrection."

The generally accepted conclusion now is that the term insanity should be substituted for *dementia* and adolescence for *præcox*. I, therefore, propose to direct your attention to-night to a few of the characteristic symptoms of adolescent insanity and I am indebted largely to Diefendorf's "Clinical Psychiatry" for my description.

Ætiology.

As already explained, *dementia præcox* is a disease of adolescence and comprises probably about 30% of all the conditions met with in patients on admission to institutions for the insane. In over 60% of patients it appears before the twenty-fifth year. Hebephrenia appears before that year in three-fourths of the patients; catatonia in 70% and the paranoid form in only about 35%. The disease in the younger patients generally seems to take the form of a simple, gradual, progressive deterioration. In cases of later onset it assumes the acute and sub-acute forms, with catatonic symptoms, while, if occurring later in life, the paranoid or delusional formation is more prominent.

Defective heredity is a very prominent factor. It varies, however, in the different forms, being far more prominent in the paranoid and less prominent in the catatonic and hebephrenic varieties. The usual physical stigmata of degeneration, such as asymmetries and malformations of the skull, ears, palate, strabismus, etc., are generally present. About 20% exhibit mental peculiarities from early youth, such as seclusiveness, affectation, eccentricity, impulsiveness and moral instability, while a small percentage of patients have been recognized to have always been weak-minded.

In women, child-bearing appears to be a very important factor, as one-fourth of the female catatonics become afflicted either during pregnancy or at child-birth. In a small percentage there is a previous history of some severe illness, from which time the patients have exhibited some change in their mental attitude. Alcoholism, as a rule, is an unimportant factor. Pregnancy favours the paranoid forms; child-birth the catatonic forms.

Pathology.

There are no pathognomonic changes to be recognized in adolescent insanity. The usual pathological evidence common to other forms of insanity may be detected. As far as our knowledge goes now, there is occasionally a reparable lesion; but more frequently the impairment and destruction of the cortical neurones is permanent and progressive.

General Symptomatology.

I propose first to deal with the characteristic symptoms met with in adolescent insanity. As a rule, there is very little disturbance in the field on apprehension. Ordinary external impressions are correctly appreciated, the patients being able to recognize their environment and to understand what takes place about them. As, however, the disease advances and mental deterioration appears, apprehension, together with other mental phenomena, are perceptibly impaired.

As a rule, orientation, especially in the hebephrenic variety, is not disturbed. However, it is common in stupor and in cases of anxiety for this to be somewhat clouded. Hallucinations, especially in the acute and sub-acute forms, are quite common and at times they persist throughout the whole course of the disease. As a rule, however, they tend to disappear in the later stages, though sometimes

they appear during exacerbations. Hallucinations of hearing are the most common, then come hallucinations of sight and taste. At first they are distressing and cause the patient much anxiety; but later they do not excite much reaction.

Consciousness in the hebephrenic variety is usually clear; but in the conditions of stupor and excitement there is at times some clouding.

There is a marked impairment of voluntary attention. Although the patients perceive objects about them correctly, they do not observe them closely or appear to understand them. In deep stupor and in advanced dementia it is impossible to attract the attention in any way. In catatonia the presence of negativism inhibits all active attention. There is a gradual and progressive impairment of memory at the onset. Memory images formed before the onset are retained with wonderful persistence; in other words, their retention is good. However, it is difficult to get the patients to reproduce these memories unless perhaps they are aroused by some unusual stimulation or excitement. Memory for events occurring after the onset of the disease is very poor; in other words, the impressibility of memory is unimpaired for past events, but greatly impaired for recent. The train of thought gradually becomes disturbed by the appearance of a characteristic looseness and desultoriness which largely influences the conduct and speech of the patient. In severe cases there is a definite confusion of thought, with marked incoherence and neologism (the invention of new words). In catatonia we come across evidence of stereotypy (the morbid persistence of a volitional impulse when once started). At times we come across a tendency to rime or repeat senseless sounds.

There is a progressive defect from the onset in judgement. The patients are unable to adapt themselves to new conditions and this condition of defective judgement becomes the basis for the development of delusions. At first these delusions are fairly stable, but later they tend to change frequently, new ailments suggested by their environment being added. The delusions, which as a rule at first are of a depressing nature, later become expansive and grandiose. In the paranoid forms there persists a wealth of delusions and, as the disease progresses, these as a rule become more and more incoherent.

A marked characteristic is the disturbance of the emotional field. This very often is the symptom which first calls attention to the approaching disease. It is noted perhaps that there is a change in disposition, a laxity in morals, a lack of affection towards relatives and particularly an abnormal satisfaction in their own ideas and behaviour. The absence of emotion becomes more marked as the disease progresses. The patients express neither joy nor sorrow, have neither desires nor fears and are quite unconcerned and apathetic, often simply gazing into space with a total disregard to their surroundings. They become regardless of their personal appearance and especially in catatonia quietly submit to being posed in the most grotesque positions. Concomitant with these emotional disturbances are found disturbances of conduct and the most prominent is the progressive disappearance of voluntary activity. The patient may probably neglect his duties and sit as described before, motionless and unoccupied for hours. It is during this stage that the patients require close observation and it is then that they are liable to perform impulsive acts, such as striking another patient, breaking windows, tearing clothing, etc..

The inability to control the impulses is very characteristic of catatonia. Each natural impulse is apparently met with and overcome by a passing impulse, giving rise to movements directly opposite to the patient's desires. In this condition, which is called negativism, the patients resist everything that is done for them, such as dressing, etc.. They refuse food which is placed before them; they refuse to open their mouth or eyes when requested or to move in any direction. This condition varies in intensity at different times and it is not uncommon for patients suddenly to assume their former activity, talking freely and attending to their own needs and then, after an interval of even a few hours, gradually to lapse back into the negativistic state. Another condition observed is the repeated occurrence of the same impulse which gives rise to what is called stereotyped movements and expressions. Patients repeat for hours the same expression or they may perform the same monotonous movement for a lengthy period, hence, what is called the verbigerations and mannerisms of *dementia præcox*.

Automatism (the performance of acts without apparent volition) is present, especially in the advanced stages. The patients are very pliable and may show echolalia (the meaningless repetition of words addressed to a patient) or echopraxia (the needless continuance of some movement initiated by another). It is common to observe that the patients perhaps laugh and cry without apparent reason. They exhibit great anxiety or become extremely irritable on the slightest provocation and, even although irritable, there is a marked discrepancy between the feelings and the facial expression. This is called paraminia (the loss of power to make natural or expressive gestures or movements). The capacity for employment is seriously impaired. The patients may be trained to perform some simple routine work; but fail utterly when given something new or complicated to do.

Classification.

There is no hard-and-fast division into groups of *dementia præcox* or adolescent insanity, but, as in general paralysis of the insane, for purposes of description three stages are recognized, so *dementia præcox* has been divided by Kraepelin into three groups, *viz.*, hebephrenic, catatonic, paranoid.

The Hebephrenic Form.

This form represents about 60% of cases of *dementia præcox*, the majority of which develope before the age of twenty-five. The first symptoms, as a rule, appear at the age of puberty; but the onset at times is so gradual that no actual date can be placed and for this reason these patients do not

come under medical observation until years after the onset of the disease. In this form there is a constantly increasing feeling of physical incapacity that varies with morbid sensations, which evidently prevent the patients from practising any sort of activity. They develope an emotional indifference and general lethargy without as a rule hallucinations or delusions. These patients generally complain of some physical trouble, such as headaches or insomnia. They lose their accustomed energy; their disposition changes; they become self-absorbed, seclusive, irritable and obstinate. They become thoughtless, irresponsible and unbalanced. They become broody, sitting about unemployed and at times take to their bed and remain there for a lengthy period. They become restless and suffer from a loss of mental application. It is at this period that many patients exhibit a very marked change in their sexual attitude. Their condition varies from time to time; but, generally speaking, there are alternating periods of depression and elation and these changes are in the female sex influenced by the menstrual period. The patient may become apprehensive, depressed and reserved, with morbid thoughts and ideas of suicide. They generally are hypochondriacal. There is a general mistrust and a feeling that they are being watched, imposed upon or conspired against. As before mentioned, it is striking to note the indifference with which the patients express their morbid ideas. In a number of patients this condition developes no further; but in some it progresses and hallucinations, mostly of an unpleasant nature, are exhibited. Gradually delusions develope and these as a rule are of a depressing character. Later these delusions may change and become expansive. At first there is considerable insight into their condition. They are conscious that a change has come over them and that they are different; but this insight, as the disease progresses, as a rule, gradually disappears. At first there is no clouding of consciousness and they are well orientated. In more pronounced cases, with hallucinations and delusions, this does not obtain and the patients are morbidly suspicious of all around them.

Early in the trouble memory is little affected; but as the disease goes on, although the impressibility of memory is retained, the patients fail to use it simply because of lack of interest. There is, therefore, an increasing limitation of thought and past experiences are not recalled and so gradually fade from the memory. Defects in judgement appear early and become more pronounced if the patients leave their usual routine life and attempt to adapt themselves to new circumstances. There is a marked evidence of emotional dulness and indifference. Their thoughts, as a rule, are very self-centred and their minds exhibit a marked lack of ballast. They go to all kinds of extremes, even in the direction of religion, sexual conduct, etc.. They lose their normal affection and are regardless of the loss of friends, visits of relatives or kindnesses bestowed upon them. They become childish and silly in their conduct, at one time very headstrong, at another time tractable. They become indifferent of their own personal appearance, perform various foolish and often serious deeds, such as breaking windows, setting fire to buildings, etc.. Their laughter is silly and unrestrained and occurs without the slightest provocation and without any emotional significance. Their conversation is childish and indicates confusion and looseness of thought and often their handwriting exhibits a carelessness and eccentricity beyond normal. As is said before, there is no hard-and-fast line between hebephrenia and catatonia. At one period the patient may exhibit the characteristics of one form and shortly afterwards exhibit the characteristics of the other.

The Catatonic Form.

This form is characterized "by stuporous states with negativism, hypersuggestibility and uniform muscular tension; excited states, with stereotypy and impulsiveness, leading in most cases, with or without remissions, to mental deterioration." This form comprises about 20% of *dementia præcox* cases.

The onset is often similar to that observed in the hebephrenic form. The patients change and become unusually quiet and serious, often complaining of a difficulty of thought and of peculiar sensations in the head. They become nervous and suffer from general ill-health. Later hallucinations and delusions appear. The hallucinations are generally of an unpleasant type, while the delusions are often of a religious nature. There is noticed particularly changes in their movements and actions, which may increase into a state of muscular tension. They assume constrained attitudes, holding the arms in awkward and often in extraordinary positions. In some there is a tendency to execute rhythmical movements, such as rolling the head from side to side. The consciousness is clouded, orientation disturbed and the patients generally do not appreciate their surroundings. Their reasoning is defective. Their memory for past events and impressibility is wonderfully good. There may be mistakes as regards identity, but the person in their mind is always remembered. In a few cases falsification of memory is seen. The emotional attitude in early stages corresponds with the nature of the delusions and hallucinations. Later the patients apparently become more accustomed and indifferent to their environment and condition and lose their early insight; their actions are not so much influenced by their delusions and hallucinations. They become listless, neglect themselves and are inclined to indulge in excesses. Gradually the process advances and catatonic stupor or excitement appears. These symptoms, however, sometimes occur in the very onset.

Catatonic stupor is chiefly influenced by negativism and automatism. Negativism often occurs first as mutism, when the patients begin by speaking lowly, breaking off in the middle of a sentence, answering in monosyllables or whispering unintelligibly and finally refusing to speak altogether. Some of these patients can be persuaded to write their answers to questions. When spoken to they often sit either with closed eyes or stare fixedly at some particular object. No external stimulus succeeds in eliciting a response. Sometimes they sit with their

lips tightly closed, but at other times they will walk away indifferently. Another example of negativism is the obstinate resistance which the patients make in an attempt to handle them. They resist being put to bed or being got up, dressing or undressing, removal from one place to another, etc.. Sometimes patients may be induced to carry out what is desired by telling them to do the opposite. Even natural impulses are resisted, such as the refusal to wear shoes, to sit on the floor rather than on a chair, to sleep under a bed and not in it. Some have preferred to eat another patient's food rather than their own or they may deliberately refuse food.

Associated with negativism is an unusual uniform muscular tension. In this condition patients may retain the same position for weeks; a very common position is for the head to be drawn back, the eyelids closed and the lips pursed together, the hands clenched, even to such an extent as to make definite marks by the tops of the fingers in the palms. In extreme cases almost absolute rigidity is seen. In this form echolalia and echopraxia are seen. The apparent opposite states of negativism and hypersuggestibility may pass directly from one to another. Some patients awaken from their stuporous condition and talk as if nothing had happened and again relapse in a few hours afterwards to their former state. Interrupting the stupor, sometimes following it and sometimes preceding it, we find catatonic excitement, which is characterized by impulsive actions and stereotypic movements. This condition usually developes rapidly. The patients will perhaps suddenly jump from their seat, cross the room, either break a window or strike another patient and then return to their original position and remain there perhaps for hours. In others the excitement is more continuous and the patients become very troublesome, untidy, mischievous and dirty in their habits. Sexual excitement is very common in this condition.

Mannerisms in speech and facial expression are very characteristic of catatonia. When speaking they make strange gesticulations, grimaces or purposeless movements of the head. The voice often is a monotonous intonation. Usually they repeat a series of senseless words over and over again and often there is a marked tendency towards riming. Their answers to questions are often irrelevant and the coining of new words is sometimes met with. In a few cases agrammatism is present, where the patient is unable to utter words in their correct sequence. Verbigeration is a common symptom in catatonic excitement as well as in stupor. This verbigeration is seen not only in the speech, but in the writings of the patients.

Although their consciousness is to some extent clouded, the patients are seldom completely disorientated and, although apparently quite unable to comprehend their surroundings, the patients, when they wake up, generally are able to give a wonderfully accurate description of the occurrences around them whilst they were in the stuporous condition. A few patients have a partial insight into their condition and will admit that their peculiar acts are foolish, but will say that they are unable to control themselves.

As previously mentioned, a patient suffering from either catatonic stupor or excitement may pass into either the hebephrenic or paranoidal form.

The Paranoidal Form.

This form tends to develope later in life than the other two forms and is characterized by persistent delusions and hallucinations, accompanied later by more or less rapid deterioration. This form has been divided into two groups.

Dementia paranoides is characterized by the persistence of many incoherent and changeable delusions ,either of a persecutory or expansive nature, or both, with slight evidences of excitement and a rapid development of dementia. These delusions are changeable and at times become very fantastic. The patients show agitation. They are anxious, restless, quarrelsome and emotional. Their conduct is often influenced by their delusions as well as their emotional attitude, but their orientation is little disturbed. They have little insight into their condition and later in the disease their consciousness becomes somewhat clouded. They are generally exalted rather than depressed. They are capable of employment, provided they are under close supervision.

The second large group is characterized by fantastic delusions, with hallucinations which are more coherently developed and expressed for a number of years, when they either become incomprehensible or disappear altogether, leaving the patient in a condition of moderate dementia. The symptoms in this group develope more gradually. The patients first exhibit despondency, with unpleasant thoughts of death or religious doubts. They accuse themselves of many wrong deeds. Gradually delusions of persecution develope and they become morbidly suspicious of others. They think that everything around them is changed; their home is unsatisfactory; the attitude of their children, etc., towards them is altered. The most common form of hallucinations is that of hearing, which developes and assists in the elaboration of the delusions. Ordinary physical sensations, such as headache, indigestion, etc., are attributed to the influence of their enemies and ideas that their food is tampered with are very common. Gradually expansive ideas are developed and these often are very fantastic. Consciousness is unimpaired, even for a long period, and they are well orientated. They have little or no insight into their condition and their emotional attitude, as a rule, is little disturbed. Their conduct is influenced considerably by their delusions; some take extraordinary steps to protect themselves from the attacks of their persecutors. Emotional mannerisms develope and in a few cases negativism and stereotypy may be seen.

Prognosis.

In all cases prognosis must be guarded. Hereditary history plays an important part, as also does the age of the incidence of the attack. Case after case will come under your notice of a neuropathic condition appearing at the age of puberty. All will

recognize that this is the time when the greatest strain is exerted on the individual. Not only are there enormous changes going on in the physical metabolism of the body, but many extraneous influences on the mind are at work. This especially obtains between the ages of twelve and twenty-five. It is during this period that the individual, both objectively and subjectively, appreciates the impressions, especially from the opposite sex. A steady progress of the developmental changes in the emotions, imagination and volition are seen. These changes require not only the inhibition of the individual, but the beneficial influence of those more experienced. It is not surprising, therefore, that we find a mental breakdown at this time of puberty, just as we commonly look for it at the climacteric. In the hebephrenic form in about 8% or 10% the symptoms disappear; but later in life there is a great risk of a recurrence followed by a definite deterioration. The patients who originally mapped out an intellectual or active business life, as a result of the psychosis, find themselves compelled to take up a calling which demands less intellectual work. A further few recover sufficiently to be discharged from the institution, provided they can be supervised by their relatives and friends. This class are unbalanced, self-occupied in reading or in pondering over useless schemes. They gradually become more circumscribed and their outlook narrowed; as a result, the prospect of their being self-supporting is poor. The large majority of patients gravitate gradually by one channel or another into a mild dementia unless cut off by some intervening disease, such as pneumonia, phthisis, etc..

In the catatonic form the usual course is that depression and stupor are followed by excitement, which eventually passes into dementia. Some patients miss the excitement. Fully 60% ultimately exhibit mental deterioration. They become stupid and lose their mental activity. Their memory becomes defective, their judgement poor and they are unable to acquire new knowledge. They are regardless of themselves or their appearance; in short, they pass into a quiet dementia. Under 20% of these patients appear to recover. Even these present slight peculiarities and changes of conduct and a number suffer from a later attack which generally terminates in dementia.

This leads me to a prominent feature in the course of this disease, *viz.*, remissions. Remissions for a few hours, days, weeks or even years are common. At these times the consciousness of the patients is clear. They understand and remember events, are quiet and rational. If, however, they are closely observed they disclose a certain restraint in manner and actions and are somewhat emotional. To the casual observer they appear to be completely recovered, yet if watched more closely peculiarities, such as irritability, seclusiveness and affected manners, may be noted.

About one-fourth of the patients regain clear consciousness, are quiet and orderly and are able to return home and resume their former occupations. They do not, however, quite resume their former status. Their judgement is slightly defective; they are somewhat listless and dull and lack energy and application. These patients often exhibit a tendency to periodic attacks of mild excitement of short duration, when they become talkative and elated, with stereotypic activity.

Somewhere about 10% appear to recover; but these patients to the close observer manifest slight peculiarities. A certain number later in life suffer from another attack.

Unfortunately, it is impossible to forecast which patients will recover, which will have remissions and which will become demented.

As a general rule it may be said that an acute development, either of stupor or excitement, is more apt to have a remission. A quick improvement, without a concomitant clearing of consciousness and a corresponding change of emotional attitude, is unfavourable. If senseless delusions, without corresponding effect on conduct, if mannerisms, etc., persist, or if there is a recurrence of excitement, the prognosis is not hopeful.

In the paranoidal forms the course is usually progressive. Dementia appears perhaps in a few months, but generally in a few years. Occasionally remissions are seen; but the outcome usually is dementia, the period of development of which varies greatly from a few months to a few years.

Diagnosis.

As already stated, there are no pathognomonic symptoms of *dementia præcox*. In fact, many of the most characteristic signs, such as negativism, automatism, stereotypy, etc., occur in other forms of mental disease. Therefore, the diagnosis must rest, as, for example, in paranoia, on the entire picture and not on one particular symptom.

Hebephrenia is distinguished from neurasthenia chiefly by the evidences of dementia, the silliness of the patients' ideas, their faulty judgement and emotional apathy, especially compared with the irritability of the neurasthenic.

Acute clouding of consciousness and speech confusion must be distinguished from *amentia*. Negativism, stereotypy and automatism indicate *dementia præcox*. The *amentia* patient is more natural, less constrained and not silly and eccentric. Further, orientation and impressibility of memory are more impaired in *amentia*. The *amentia* patient endeavours to concentrate and reply properly to questions, whereas the *dementia præcox* patient answers either in a silly manner or not at all. In *amentia* the emotional attitude is very changeable; but in *dementia præcox* it exhibits stolidity and apathy.

Early catatonia may be mistaken for "epileptic befogged states." The negativism of the catatonic is distinguished from the anxious resistance of the epileptic. The catatonic is better orientated. Epileptics are more anxious and are more liable to make deliberate assaults and attempts to escape, whereas the catatonic impulsive acts are more mischievous and purposeless.

To distinguish the depression of *folie circulaire* from that seen in hebephrenia and catatonia is often difficult. Early hallucinations, foolish delusions, especially of a somatic nature, and emotional atti-

tude not corresponding to the delusions or surroundings indicate *dementia præcox*. Hypersuggestibility is more pronounced in *dementia præcox*. In the negativism of the catatonic there is uniform, rigid and even stubborn resistance to every passive movement, whereas in ordinary melancholia passive movements are permitted, though the patient exhibits resistance if threatened with injury and attempts to protect himself. In catatonia voluntary movements are rare; but if they occur they are carried out rapidly, except when made by request, when there is generally delay. In melancholia all movements are slow.

In the excitement of catatonia the clouding of consciousness is less marked than in mania and the patient better orientated. The speech of a mild catatonic is more senseless than even that of a severe maniac and the former indulges in verbigerations and stereotyped expressions, whereas the maniacal patient discourses flightily more on his immediate surroundings and is more easily distracted. Catatonic movements are purposeless and silly; maniacal movements purposeful, suggested by the surroundings and immediate impressions and emotions. The activity of a catatonic is generally limited to a bed or one portion of a room, whereas a maniac goes all over the place.

The paranoidal forms of *dementia præcox* are distinguished from paranoia by the lack of system, the rapid development of fantastic delusions, with pronounced hallucinations. In paranoia the onset is very gradual and as a rule hallucinations are not a prominent feature. The *dementia præcox* delusions are more fantastic, more changeable, less systematized and do not fit in, as a rule, with events occurring in the patient's life. Paranoia delusions generally are morbid interpretations of actual events woven together into a systematized whole and extended to include even events of recent date and the patients are more reasoning and explanatory. The emotional attitude of *dementia præcox* is more changed. In paranoia the demeanour is more natural and the patients are less self-centred and more employable.

Treatment.

Of all forms of insanity there are none which open a greater field for thought, which offer a greater field for research and which, in my opinion, hold out greater prospect for success than *dementia præcox*. This form of insanity is attracting more and more attention amongst alienists, so much so that in Chicago there is even a society for the promotion of the study of *dementia præcox* which publishes a journal called *Dementia Præcox Studies*, which is always full of interest. In countries more favourably situated than Western Australia, where there are up-to-date laboratories, whose staff are able to devote their whole time to research, excellent results have been obtained from the bacteriological examination of patients and their subsequent treatment by vaccines and sera.

Our present knowledge of the causes of *dementia præcox* is so scanty that, generally speaking, our treatment is restricted to the treatment of the various symptoms. Excitement, insomnia, defects in nutrition, etc., are treated in the usual way. Last year Dr. Bentley directed your attention to cases of ductless gland therapy in the insane. There is absolutely no doubt that the mental condition of a patient in very many cases can be changed at will by the exhibition of extracts of one or other of the ductless glands. A few examples will be shown you to-night. The treatment of stupor by thyreoid extract has long been practised, with excellent results in many cases. We all know the effect of thyreoid on cretinism and all are agreed that the effect is entirely dependent on the age at which the treatment is started. Many of our disappointments in the treatment of *dementia præcox* by ductless gland extracts are doubtless for the same reason due to the lateness at which treatment is started. I am hopeful that, as we acquire greater knowledge of this comparatively recent therapy and as we become enabled to recognize more definitely the prodromal symptoms of *dementia præcox*, our recovery rate will increase.

Gentlemen, to-night I have endeavoured, in the limited time at my disposal, to present to you a clinical picture of *dementia præcox*. In doing so I candidly admit that my remarks are not original and that I have freely utilized the brains of others, especially Diefendorf.

You may think that ductless gland therapy is almost an obsession with us at Claremont, but I can assure you that the results we are getting are sufficient to justify us in urging you all to make it your special study and I forecast that, if you do, it will be of benefit, not only to yourselves, but to the public in general.

THE PRESENT PLAGUE OUTBREAK IN SYDNEY.[1]

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THE present invasion of Sydney by plague has exhibited some features of peculiar interest. Within a small compass it has presented a very perfect picture, both epidemiologically and epizoologically, of a typical plague outbreak and, largely owing to its hitherto limited extent, its observation was able to be very thorough. The links in the chain of evidence illustrating the progress of the rat epizootic have been fairly complete and the connexion between each human patient and the infected rat has, with few exceptions, been established. Additionally, and this is an unusual feature of plague outbreaks, the very earliest beginnings of the epizootic were observed and traced with considerable certainty to the actual ship which was responsible for the introduction of the infection to Sydney from overseas. On the therapeutical side the outbreak has witnessed the introduction and use of what appears to have proved to be an effective curative serum.

The Commencement of the Epizootic.

The first intimation to this State that bubonic plague had once more invaded Australia was a

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 7, 1922.

telegram published in the Sydney morning papers on September 15, 1921, which announced that a death from plague had occurred in Brisbane on August 23, 1921. On the same date a formal communication confirming the press announcement was received by my department from the Department of Health of the Commonwealth.

No other developments occurred until September 19, 1921. On that date the steamer *Wyreema* arrived from Brisbane in the early morning and, after examination by the Federal Quarantine authorities, was allowed to come alongside No. 2 A.U.S.N. Wharf and began discharging her cargo under supervision of a Quarantine Officer. In the afternoon six dead rats were found beneath the cargo in No. 4 hold of the vessel. The Quarantine Officer in charge immediately caused them to be taken to my department, where, on bacteriological examination, one was found to be plague infected. The others had probably died of plague also, but were too putrid for examination.

The Quarantine authorities were immediately informed and the holds of the *Wyreema* were closed and fumigated by them. Early next morning the *Wyreema* was removed from the wharf and anchored in the stream under control of the Federal Department of Health. On September 20 a large number of rats found on board were sent to the Department for examination and eleven of them were found to have plague. All the rats sent from the *Wyreema* were of the black variety (*Rattus rattus*).

On September 21, 1921, two black rats were found dead on the A.U.S.N. Wharf, near where the *Wyreema* had been tied up. On examination they were found to be plague infected.

On September 22, 1921, a grey rat (*Rattus norvegicus*), caught on the same wharf, proved on examination to be plague infected and between that date and October 10 twelve more infected rats were found, either upon the same wharf or in an engineering workshop situated at the base of the wharf, during operations of cleansing and disinfesting which were carried out at the wharf throughout the period in question.

All the dead rats found in the process of cleansing this locality were recently dead. Only two of them were too decomposed for bacteriological examination and they were found towards the close of the cleansing operations. Such an absence of decomposed carcases is inconceivable in places where plague has been prevalent for an appreciable time among the rat population and indicates that plague had invaded the wharf contemporaneously or nearly so with the first discovery of plague rats.

Another important fact is that a particularly strict watch was always kept on this wharf, owing to its special susceptibility to plague invasion. Between August 1 and September 20, 1921, twenty-seven rats had been caught there upon nineteen different dates. All of these rats were healthy. No plague rats were found upon the wharf until after the arrival of the *Wyreema*.

In order to follow more correctly the progress of the rat epizootic and to obtain a better grasp of the whole outbreak, it will be convenient to deal with it in four groups or areas which I have named the north-central, the south-central, the Paddington and the Woolloomooloo areas. Though these areas or groups are quite distinct upon the map, they have certain relations to one another which will presently appear. This method of description involves a little overlapping in the time relation, which is, however, of no importance. The epizootic in each area, with its accompanying human infections, constitutes a group of facts complete in itself.

The Spread of Infection.

The sequence of events in the north-central area was, as is evident from the map, quite simple. A small, compact group of seven business premises situated in Sussex Street and Day Street, in the immediate vicinity of the A.U.S.N. Wharf, and one outlying building in Kent Street, about one hundred and fifty yards distant from the main group, had been invaded by plague rats from the wharf before the advance of the epizootic in this neighbourhood could be checked. Although thirty-eight infected rats in all were taken in this area, only one human infection occurred—at No. 102, Sussex Street, on December 3 (Case No. 2). The last plague rat in the area was taken on January 23, 1922.

The south-central infected area is nearly half a mile distant in a southerly direction from the north-central area and, although the district between the two areas has been assiduously searched and trapped from the first beginnings of the outbreak, no plague rats have been taken there. In contrast to the compactness of the northern area, the infected premises in this group are strung out in a straggling line, stretching east and west from Sussex Street to Castlereagh Street. This area yielded twenty-three plague rats from four premises (besides a number of carcases presumably dead of plague, but too putrid for examination), together with four infections in human beings. The evidence as regards the localization of the infection of one case, however, is presumptive rather than absolute.

Plague rats were first found in this area in a large produce store at 361, Sussex Street, on November 1 by the City Council rat staff. Fifteen plague rats were obtained on these premises, but no human infection occurred. The next door premises, however, yielded the first human patient of the outbreak—on November 26, 1921. Though no proved plague rats were discovered here, two or three carcases of rats, which were suspected of having died of plague, but were too putrid for examination, were found on the premises. They had been no doubt infected from next door.

The manner in which plague infection reached the rat population of No. 361, Sussex Street, could not be ascertained with certainty. Careful investigation failed to disclose any opportunities for the conveyance of rats from the A.U.S.N. Wharf or the north-central area. It was considered not improbable that infection was introduced directly from Brisbane in cases of eggs which were regularly received by the firm occupying the premises, from the Brisbane branch of their business, in connexion with which the first Queensland case of plague had oc-

curred. On the other hand, one cannot negative the possibility of the infection having been brought to the store from the A.U.S.N. Wharf after the arrival of the *Wyreema*.

One of the human cases connected with this area was surrounded by circumstances of sufficient interest to warrant a detailed statement of the facts here:

On February 9, 1922, a schoolgirl (J.L., Case No. 4), aged thirteen, died rather suddenly at a boarding-school in Parramatta. She had been ailing since the afternoon of February 5, but her illness did not at first appear to be serious. On the seventh and eighth she became worse and vomiting set in. On the morning of the ninth the medical practitioners in attendance suspected plague and communicated with the Department. Dr. Robert Dick, of the Health Department, visited Parramatta, but the girl was dead before he arrived. He made a *post mortem* examination and the diagnosis of septicæmic plague was verified bacteriologically.

How had she been infected? As far as was known, Parramatta was entirely free from plague infection and has remained so right up to the present time. A very careful search of the school buildings disclosed no evidences whatever of the presence of rats and the girl had not been away from the school since she returned there on January 29 after the summer holidays. She had spent the holidays in question with an aunt at Brighton-le-Sands. This latter district was also free from all suspicion of being infected and the aunt's premises were on inspection found to be free from rats. On January 24, however, the girl had been to Sydney with her aunt and had visited several shops in the Liverpool Street block of the south-central infected area and had in particular spent over an hour making purchases of underclothing in one large drapery establishment in the block. There must have been plague rats in the block on January 24, when J.L. visited it, as, four days later, on January 28, a human case of plague occurred in the block and several plague rats were taken there, including, at a later date, one in the actual drapery establishment visited by J.L..

It will be observed that a period of twelve days elapsed between the time at which the patient visited the infected area and the date of her attack. This is a long incubation period for plague, which does not as a rule exceed five days. The Indian Plague Commission, however, ascertained experimentally that a plague flea might under certain circumstances remain infective for fifteen days after it had left its rat host. The possibility, therefore, cannot be overlooked of J.L. having picked up a rat flea in the course of her visits to shops in the infected area and having been bitten by it at a later date. Such an occurrence, though an evident possibility, is unusual and the course of infection in this case must still be regarded as obscure. After careful investigation, however, it is the only likely hypothesis which can be advanced.

The Paddington area. On February 18, 1922, the Medical Superintendent of the Royal Prince Alfred Hospital reported the case of J.A.R. (Case No. 5), a man aged seventeen, residing in Comber Street, Paddington, and employed in a produce store situated at 76, Oxford Street, a few yards from his residence, as one of plague. The patient was immediately removed by ambulance to the Coast Hospital, where the diagnosis was confirmed. The produce store was quarantined and searched for rats. Two carcases were found, but both were too decomposed for bacteriological examination. There was, however, good reason for believing that they had died of plague. A history of sick rats having been seen about the store was also obtained. When admitted to the Coast Hospital, J.A.R. was very ill and delirious, but a few days later he stated that he had seen dead rats in the basement of the store where he had been working a few days before his illness. Examination of the store disclosed numerous rat holes and abundant traces of rat infestation. On investigation it was ascertained that several loads of chaff and other produce had been brought to this store from a plague-infected establishment in Sussex Street (north-central area) between three weeks and a month before J.A.R. was attacked and it seemed clear that a plague rat must have been concealed in the produce and infected the rats in the Oxford Street store.

The area of Paddington in which this produce store is situated, was in a bad condition structurally. The buildings upon it facing Oxford Street are for the most part small, combined shops and dwellings and at their rear is a closely packed mass of old, small terrace dwellings crowded between narrow lanes. In many cases the yards at the rear of these premises had been wholly or partially covered by stables and sheds of the roughest description, quite unsuitable for a modern city and affording abundant harbourage to rats, which was enhanced by considerable accummulations of rubbish. Similar harbourage was disclosed in most of the houses, which were generally in a bad state of repair. As a matter of fact, the whole area proved to be rat infested to a marked degree. It was clear that the opportunities for the spread of plague from the rats at the produce store were numerous and a strong staff of rat-catchers was turned into the area to search it and, if possible, to clear it of rats. The search was rewarded by the discovery of plague rats on several of the immediately surrounding premises, including Nos. 72A, 80, 84 and 88, Oxford Street, and No. 5, West Street, while several carcases of rats, which were ascertained to have died of plague, were picked up in the streets and lanes of the area.

Two other human cases of plague occurred in this area (Cases Nos. 7 and 8). Both the patients acquired infection at No. 84, Oxford Street, while the premises were in quarantine on account of a plague-infected cat and a plague rat having been found there. One was the wife of the occupier and the other a young tradesman engaged in carrying out repairs to the premises enforced upon the owner by the Department. This young man's case is specially interesting from the fact that the incubation period of his attack could be very accurately fixed. He began work on these premises on the morning of March 7 and continued there during that day. On the eighth he was not at the premises at all, being

engaged at work in another district. On the ninth he again returned to 84, Oxford Street, and worked there till 4.30 p.m., when he returned to his home at Mosman and was taken ill with a headache and rigor at 9 p.m.. He must have been infected on the seventh, between 10 a.m. and 5 p.m.. The incubation period in his case was therefore more than fifty-two hours and less than fifty-nine hours.

The Woolloomooloo area. The Woolloomooloo group of cases or sub-epidemic presents many interesting features. It centred round a livery stable with dwelling attached, situated at 119, Dowling Street, in a residential district. On March 22, 1922, a motor-car driver (J.C., Case No. 9), a single man, residing in lodgings in Darlinghurst and employed in a motor garage in Castlereagh Street, died suddenly of plague in the Sydney Hospital before he could be removed to the Coast Hospital. Careful investigation led to the conclusion that he had not contracted infection either at his lodgings or the place of his employment and for a few days the Department was at fault. At length, a minute inquiry into his movements during the week before his illness disclosed that on at least two occasions during that time he had spent the evening at 119, Dowling Street, Woolloomooloo, and that a young woman residing on the same premises had recently had an illness described as "slight" from which she was convalescent. She was visited by a medical officer of the Department and the opinion formed that she had undergone a mild attack of plague, from which she had scarcely recovered, as there was still an indurated and slightly enlarged glandular mass in the right groin. The diagnosis was subsequently confirmed serologically (E.S., Case No. 14). Inquiry elicited from the convalescent herself and from other inmates of the premises that rats had been dying in the livery stables for two or three weeks before E.S. became ill. The fact of the visits of the motor driver J.C. to the premises in the three or four days immediately preceding his illness was also established and it became clear that he had contracted infection there.

On March 28 a little girl (M.W., Case No. 10) was found to be ill with plague at No. 115, Dowling Street, and it was ascertained that she had been a frequent visitor at No. 119 (two doors from her own residence) and had waited on Miss E.S. during the latter's illness. M.W. had a very severe attack of plague. The next day a stableman (R.R.) employed in the livery stable attached to No. 119 presented himself with an "abscess" in the groin, which he had had for some days and which had followed an illness accompanied by headache, shivering and vomiting. The illness, he said, had only lasted for one day. He was sent to the Coast Hospital as a plague suspect and later the diagnosis of plague was confirmed bacteriologically and serologically (Case No. 13). This case and that of E.S. are interesting as being the first "mild" cases of the present plague outbreak. It will have been gathered that four cases of plague were traced to No. 119, Dowling Street, three of which occurred before any suspicions were directed to the premises. As soon as the premises became suspect they were quarantined, searched for rats and disinfested. A number of rat carcases, all of them two or three weeks old and too far advanced in putrefaction for bacteriological examination, were found in the basement of the dwelling and the attached stables. They had evidently died of plague. The fodder used in the stables had been obtained from several firms in Sussex Street and had no doubt been the vehicle of infection to the premises. More definite information on this point was lacking. During the process of cleaning up and disinfesting the block of buildings in which No. 119 was situated, a mother and her little daughter, residing in Judge Street, in the same block as 119, Dowling Street, and at the rear of the latter, were both attacked by plague and both were very seriously ill (Cases Nos. 12 and 15). Altogether, six persons were attacked by plague in this area.

Outside of the four areas described, two cases of plague in human beings came to notice in Alexandria, an industrial suburb of Sydney, on March 29 and April 3, 1922. They are included within the purview of this paper, but no area has been set aside for them, because no plague rats have hitherto been found in that district. They are still being searched for. Including these cases, the total number of human beings attacked to date has been sixteen.

Administrative Measures.

The problem of plague in the seaport towns of Australia cannot be regarded as a passing one. Communications with eastern ports, which are often infected by plague, have become so frequent and rapid that Australia will, at any rate for many years to come, be liable to invasion and the past experiences of both New South Wales and Queensland have demonstrated that quarantine measures are unable to secure our ports from invasion without imposing upon commerce such stringent precautions as would seriously interfere with trade. It is possible that in the future an international agreement requiring the fumigation of all sea-going ships before loading and their practically entire clearance of rats by this means may in time modify the risk; but those days are yet to come.

Omitting for this occasion the question of the introduction of plague from overseas, which, as coming under the control of the Federal Department of Health, may be considered outside the scope of this brief survey, the administrative control of plague may be considered from two aspects, those of preparation and of active defence. In both directions we are fortunate enough to be the inheritors of the code of fundamental principles first laid down by that masterly epidemiologist, Ashburton Thompson.

Plague is a disease of the rat which secondarily affects man and is conveyed from rat to man by the flea. In that sentence is embodied the whole theory of plague prevention. The disease is not directly communicable from man to man, except in the case of that form known as pneumonic plague, which is rare in civilized communities. The truth of this general statement is illustrated by the fact that during the whole of the nine years (1900-1909) in which plague was prevalent in Sydney no hospital nurse or attendant contracted plague.

From the fundamental principle just laid down, it follows that the campaign against plague resolves itself into a campaign against the rat, which has now rightly come to be regarded as the worst enemy of mankind known among mammalian animals. He is so bold, so adaptable and so prolific that the task of exterminating him in any ordinary city is almost Herculean in its magnitude, but there is little doubt, that, by the proper construction of buildings, he will be entirely banished from the model cities of the future. In the campaign against the rat biology has hitherto failed us. Science has long sought and is still seeking some disease which, while harmless to man, will exterminate rats, but the search has so far been fruitless. Many rat viruses have been put on the market, but they have failed everywhere, including Sydney, where they have been tried on a fairly large scale on at least three occasions. In view of the fact that plague itself, probably the most fatal disease known among rats, never succeeds in exterminating them in a given district, the outlook in this particular direction does not appear very hopeful.

Apart from the regular destruction of rats, which should continue at all times in every seaport, irrespective of the presence or absence of plague, there are only two specific anti-rat measures which need mentioning here as necessary during non-epidemic times. They are the rat-proofing of wharves and buildings and the maintenance of a detective rat-catching staff, whose duties are, not to exterminate rats, but to keep going a continuous nightly trapping of the area which in a seaport town is certain to be the first attacked, *id est*, the water front. This measure will usually give the authorities the very earliest warning of an invasion of plague.

A very careful watch upon the health of the Sydney rats has been maintained year by year by the Health Department, ever since the second outbreak of plague in the year 1903. Two intelligence staffs of rat-catchers have been constantly maintained in operation since April 1 of that year. One of these staffs has been employed directly by the Health Department to catch rats on the wharves and water frontages; the other staff has been employed by the City Council and works throughout the whole of the city. Every rat caught by these two staffs is always labelled with the address of the premises on which it was found and is brought to the Microbiological Laboratory of the Health Department, where it is dissected and examined. Since 1903 over 330,000 rats have been caught by these staffs and examined as I have indicated. Full records of these examinations are always kept.

The rat-proofing of buildings and wharves is unquestionably the most important weapon in the anti-plague armoury. No city would ever be attacked by plague in which all the buildings were constructed on rat-proof principles, provided thereafter that ordinary care be taken to prevent rats from breeding in them. Unfortunately, the reconstruction of a city on rat-proof lines is a slow and tedious undertaking and though a majority of the buildings in the business centre of Sydney between Darling Harbour and the line of the parks have been made rat-proof during the past twenty years, there remain too many which have not yet been brought up to date in this respect. As far as the wharves are concerned, the work done by the Harbour Trust during the same period has been very satisfactory. Over six miles of wharfages, including nearly the whole of those used by deep-sea and inter-State shipping have been faced with concrete sheeting and practically all store-houses and other buildings on the wharves or abutting on them have been floored with the same material and otherwise rendered as nearly as possible rat-proof. The City Council and some of the suburban councils have also been active in the direction of enforcing rat-proof construction in produce stores, stables and some other classes of buildings specially liable to rat infestation and, though several of the suburban councils have entirely failed to realize their duty in this direction, there is no doubt that Sydney is in a far safer position with respect to plague than it was in 1909, when the last outbreak ended. These structural improvements greatly facilitated the handling of the present outbreak and have been largely responsible for its limitation within very narrow bounds.

As soon as the announcement of the invasion of Queensland by plague was made in September, 1921, the rat intelligence staffs were largely increased in personnel and their operations were practically confined to the threatened area—the water front and that portion of the city situated between George Street and Darling Harbour—and certain portions of that area were worked with special intensity. As the epizootic extended the dispositions of these staffs were modified from time to time as requisite.

Wherever a plague rat is found or a case of human plague occurs, the premises on which the rat is found or which are adjudged to have been the source of infection for the patient, are quarantined and "disinfested." The "quarantining" is not very stringent in character and simply consists in closing up the premises so as to prevent the escape of rats and forbidding access by the public until the process of disinfestation is complete. The removal of goods from the premises during the same period is also stopped. This process of disinfestation has for its object the destruction of rats and fleas and may take several days or a few hours, according to the method employed. Meanwhile, an area comprising several street blocks surrounding the infected premises is intensively searched, poisoned and trapped for rats by the official staffs. There is no question of driving abroad or scattering the rats by these proceedings. Such a result might arise in the open country, but it cannot take place in a city where the whole of the area treated is covered closely with buildings.

Two methods of freeing from plague premises ascertained to be plague infected are used in Sydney. One of these is by means of fumigation with hydrocyanic acid gas. The other consists in turning over all the stock and movable materials on the premises by the manual labour of a gang of workmen and searching out the rats, which are destroyed as discovered. The most speedy, convenient and thorough of these methods is the former. The gas makes its way into rat holes and crannies, finds out the rats

and destroys them promptly. It also enjoys the immense advantage of killing off the fleas which infest the premises. On every occasion on which it has been employed, control captive rats or guinea-pigs have been used to test the efficacy of the fumigation. Rats or guinea-pigs in cages are placed before fumigation among the produce or other goods under conditions resembling as closely as possible those which would occur naturally. That is to say, the cages are placed deeply in cavities excavated in the mass of the material stored and are then carefully enclosed by packed layers of the material. After the fumigation is completed the cages are uncovered to ascertain if the rats are killed. In the earlier experimental fumigations live fleas were also used in test tubes plugged with an inch of cotton wool. On each occasion both rats and fleas were all killed by the action of the gas. Unfortunately, fumigation with hydrocyanic acid gas is not always practicable, owing to its rapidly fatal effects upon human beings if inhaled. Buildings in which it is used must be well isolated from other buildings, either by construction or position. When it can be utilized, it is ideal for the purpose and stores containing as much as 700,000 cubic feet of space have been quickly and thoroughly freed from rats and fleas by this means. In this process one gramme of cyanide of potash and one cubic centimetre of sulphuric acid are required for every cubic metre of space to be fumigated (*id est*, ten ounces of cyanide, ten fluid ounces of acid to every thousand cubic feet). The process is not to be relied on to kill weevils, but is rapidly fatal to all other forms of insect life.

The other method of disinfesting a rat-infested building, by turning over all the contents is much slower than that by fumigation. In a large produce store or warehouse it may take days or even weeks, as against, say, twelve hours for the fumigation method. The inferiority of this method as regards the destruction of possibly infected fleas is manifest and is not entirely compensated for by the careful spraying of floors and all surfaces with kerosene emulsion, which is always done before and during any clearing up operations in a plague-infected building.

Whichever method is used, the premises are not released from quarantine until all defects in the building are made good by the owner—rat holes filled up, basements concreted and the premises put generally into a rat-proof condition.

Guinea-pig "sentinels" have been regularly used in Sydney during the outbreak to ascertain whether buildings are free of plague fleas. When used for this purpose, they are placed in stores or buildings which are known to have been plague infected and have been cleaned up and freed from rats. If plague fleas are still in the building they find their way to the guinea-pig and infect him with plague; in such a case the building is again sprayed with kerosene emulsion and retained in quarantine until sentinels have remained in the building uninfected for forty-eight hours. It is not our practice to use sentinel guinea-pigs in buildings which have been "disinfested" wth hydrocyanic acid gas, because this gas, when correctly used, not only destroys all the rats, but also the fleas and therefore does away with all danger. Guinea-pig sentinels are also stationed in the buildings immediately surrounding one which has been ascertained to be plague infected and if one of these sentinels becomes infected, measures are taken to "disinfest" the building he was placed in, exactly as would be done if a plague rat had been found there.

The method of dealing with plague patients which has been adopted in Sydney, is simple and gives rise to no difficulties. All patients are treated at the Coast Hospital. At departmental headquarters there are always available one or more medical officers with plague experience who are prepared to visit patients suspected of having plague and to consult with their medical attendants. If, after consultation, the case is considered to be suspicious of plague, an ambulance is at once sent to the patient's residence to convey him to the Coast Hospital, where, on his reception, he receives a dose of anti-plague serum. If the patient is already in a public hospital when notified to the Department or if the medical attendant is definitely of opinion that the case is one of plague, the visit from a departmental consultant is often dispensed with, on the grounds that it consumes time, and an ambulance is sent for him immediately. After the patient has been removed to hospital, the treatment of his residence and its inmates is the next consideration. In regard to the inmates, all that is done is to take their names and addresses and warn them to communicate immediately with departmental headquarters if they should become ill. If there are grounds for believing that the patient was infected at his residence or if on examination it is found to be rat infested, it is submitted to one or other of the processes of disinfestation previously described in this paper. If, on the contrary, the infection is judged to have been taken elsewhere, the residence is merely sprayed with some disinfectant preparation. No further action is taken or necessary.

The anti-plague serum referred to is a local production, manufactured in Sydney by the Biological Institute of Australia and prepared under the superintendence of Dr. Burton Bradley, formerly Assistant Bacteriologist of the New South Wales Department of Public Health. The method of preparation I shall not refer to. It will, I have no doubt, be described later on by Dr. Ferguson and the manner of its use and the results of its exhibition dealt with by Dr. Millard. It is sufficient for me to say it has been used upon every plague patient treated at the Coast Hospital in this outbreak, with one exception, and with very satisfactory results. The total number of plague patients during the outbreak has been sixteen. Of these, two died before they could be taken to the Coast Hospital. A third only reached the hospital in a moribund condition and expired two hours after his admission. In a fourth patient the illness was mild in type and was undiagnosed until convalescence had occurred and therefore the patient was not admitted to hospital. Of the remaining twelve patients admitted to the hospital, one had a quite mild infection and did not receive any serum treatment. Ten patients treated with the serum recovered and one died. The last men-

tioned was the first human being attacked by plague in the outbreak. When he was admitted to the hospital the serum was not immediately available and he did not receive a dose until the fifth day of his illness. The total mortality of the outbreak up to the present has been 25% and the total mortality among the patients admitted to the Coast Hospital 15.4% or, omitting the patient who died within two hours after admission, 8.3%. In the opinion of the medical officers of the staff who had experience of the plague epidemic of 1900-1909, the type of the disease in the present outbreak is of equal severity to that of the former one.

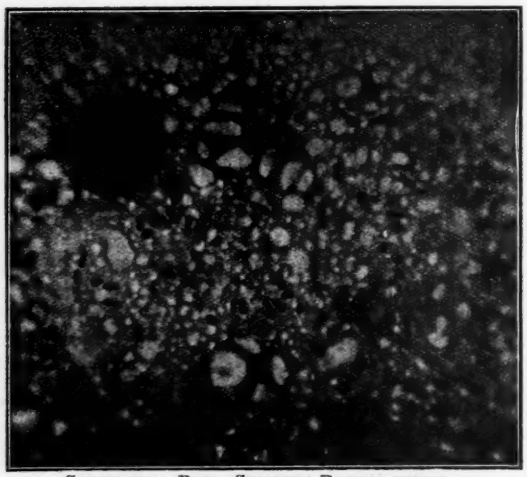
In the first outbreak of plague in Sydney in the year 1900, the mortality of the whole outbreak, in which 303 persons were attacked, was 34% or, omitting Chinese, 32.4%. The mortality among the patients admitted to hospital was 24%. Of these patients admitted to hospital, seventy-one were treated with the Roux-Yersin serum, among whom the mortality was also 24%.

THE PLAGUE EPIDEMIC.[1]

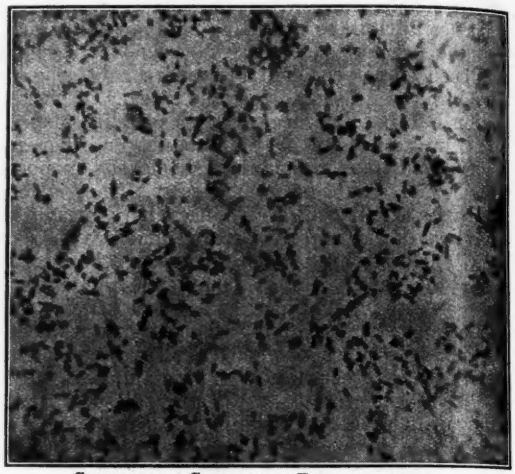
BY E. W. FERGUSON, M.B., CH.M. (SYD.),
Principal Microbiologist, Department of Public Health, New South Wales.

IN joining in the discussion on the outbreak of plague in Sydney, I wish to confine my remarks to the bacteriological aspect of the disease. In the presence of an outbreak, the bacteriologist has two main functions to perform. Firstly, he has to confirm the clinical diagnosis of the disease in human patients; secondly, he has to find infected rats. *Bacillus pestis*, the cause of plague, is a small, oval bacillus, showing marked bipolar staining. It generally can be found in large numbers in smears from buboes or in blood films from cases of septicæmia. In cultures it is highly pleomorphic, involution forms of varying shapes occurring. In human cases of *pestis bubonica* the bacilli can generally be demonstrated in smears of fluid obtained by acute puncture of the bubo, while cultures may also be obtained by the same method and animal inoculations carried out. In the present series there were fifteen patients up to the end of March. *Bacillus pestis* has been demonstrated in smears or cultures from all except two. These findings were confirmed by animal inoculation from five. In two instances the organisms were obtained *post mortem*, both from the bubo and from the spleen; these patients died from a terminal septicæmia.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 7, 1922.



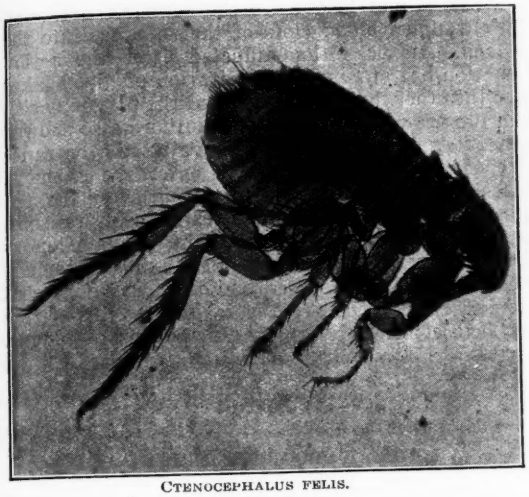
SMEAR FROM BUBO, SHOWING BACILLI PESTIS.



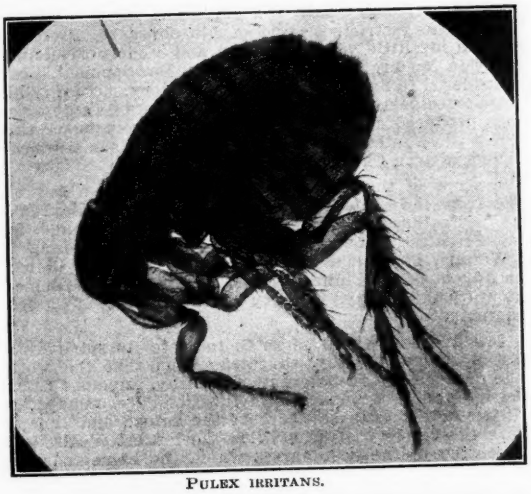
SMEAR FROM CULTURE OF BACILLUS PESTIS.

Serological tests have been carried out in six patients. In four instances the blood serum of the patient agglutinated the *Bacillus pestis* in a dilution of one in ten, though the agglutination was slow and did not take place at times until four hours. Four controls remained unaltered, no clumping being observed after several hours. The agglutination test for plague cannot be depended upon as a routine procedure; positive results are of value, but the absence of agglutination has no significance.

In rats the disease takes the form of a septicæmia, with or without the formation of an initial bubo. In the routine examination of rats the animals are opened along the median ventral line and the liver and spleen inspected. If normal, the rat is not further examined. In infected rats the liver is enlarged and congested and has always a granular appearance. The spleen is enlarged and turgid and in the more sub-acute or chronic forms miliary areas of necrosis appear in the spleen and to a less extent in the liver. These are more evident in inoculated animals. Buboes are seldom searched for in the rats examined, though they are stated by the Indian Plague Commission to be present in 85% and to be most frequently situated in the neck region. General congestion is present and there may be hæmorrhages in the lungs, intestines, suprarenals or elsewhere. Smears from the liver and spleen generally teem with typical bipolar bacilli, but in the less acute infections these may be scantier and recourse

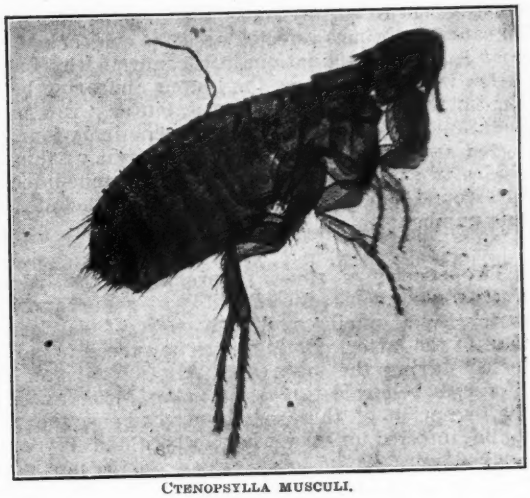


Ctenocephalus felis.

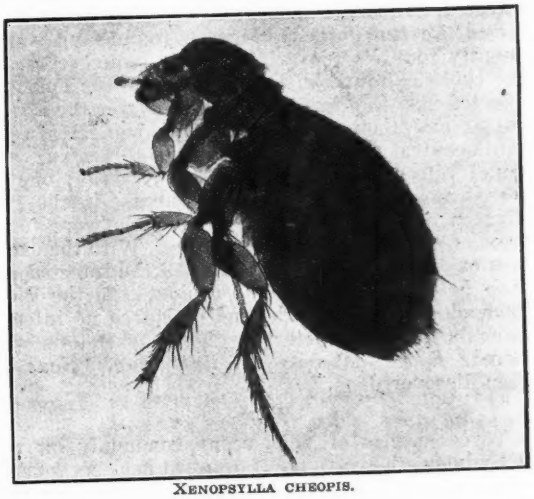


Pulex irritans.

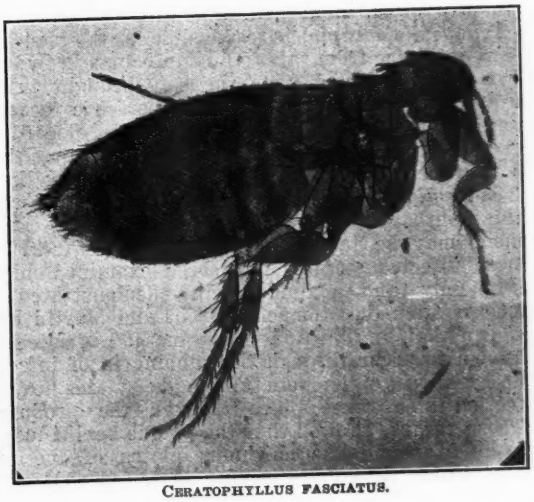
Proventricular Valve of Ceratophyllus fasciatus.



Ctenopsylla musculi.



Xenopsylla cheopis.



Ceratophyllus fasciatus.

must be had to culture for confirming the diagnosis. In rats from a new infected area the evidence of the *post mortem* examinations are confirmed by inoculation into a guinea-pig, the lesions appearing being similar to those found in the rat, but as a rule more definite. Up to the end of March ninety-four infected animals had been found in Sydney; this includes two mice and two cats. The plague-infected rats found on the *Wyreema* are also included in this number.

Rodents.

Two species of rats are prominent in Sydney, *Rattus norvegicus* and *Rattus rattus* in approximately equal numbers, slightly more of the former than of the latter. Both species have been found infected during the outbreak. All the rats on the *Wyreema* belonged to *Rattus rattus*, whereas, with the exception of three of the early ones, the rats found infected on the wharf where the *Wyreema* berthed were *Rattus norvegicus*. In the north-central area, excluding the A.U.S.N. Wharf, fifteen *Rattus norvegicus* and six *Rattus rattus* were found infected. In one building, 52, Day Street, ten infected *Rattus norvegicus* were found and subsequently four *Rattus rattus*. This sequence recalls the evidence found in Bombay, where the epizootic affected first the *Rattus norvegicus*, later the *Rattus rattus* and then spread to human beings. In the south-central area only two *Rattus norvegicus* were found infected and twenty *Rattus rattus*; one infected mouse was found in this area. In the Paddington area sixteen infected animals were found from or adjacent to seven premises. With the exception of one mouse and two cats, all the infected animals belonged to *Rattus norvegicus*. In the Woolloomooloo area up to the end of March no infected animals had been detected, though skeletons were found. Since that date one *Rattus norvegicus* has been discovered.

Fleas.

Three species of fleas occur commonly on rats in Sydney—these are the Indian rat flea (*Xenopsylla cheopis*), the European rat flea and the mouse flea; the cat and dog fleas and the human flea are occasionally found. In all probability the Indian rat flea (*Xenopsylla cheopis*) is responsible for the transmission of plague from rats to human beings in Sydney. Fleas are collected from the rats brought in for examination and the weekly numbers are recorded according to the different species found. Records of examinations of fleas made in the Laboratory show a very decided seasonal distribution, especially of *Xenopsylla cheopis*; this flea is most abundant in the months of February and March, though specimens may be taken throughout the year. Fleas from infected animals have been examined microscopically and plague bacilli demonstrated in smears from the stomach. Bacot and Martin have shown that, in addition to the method of infection known to occur through the rubbing in of infected flea fæces into the puncture made by the flea, infection also takes place through the proboscis. These authors showed that the plague bacilli lodged in and around the chitinous plates of the proventricular valve, multiplying there, as well as in the stomach and leading to the complete occlusion with entangled blood clots of the entrance into the stomach. In fleas thus affected after the action of suction has stopped, regurgitation of blood takes place into the wound, carrying with it plague bacilli from the pro-ventriculus. These authors also showed that, after a time, the clot plugging the entrance softened and allowed blood to enter the stomach, while it also permitted regurgitation after the act. Infected fleas have been found capable of transmitting infection up to fifteen to twenty days after becoming infected. The specimens shown include smears from human cases and from rats, infected rats and guinea-pigs and the various species of rats and fleas concerned in the transmission of plague.

Reviews.

MEDICAL PROGRESS IN CHINA.

IT must be rare for an author of a book which purports to subserve two distinct objects, to have fulfilled both.

Within the restricted scope of his objective, Professor Balme, in his little volume, "China and Modern Medicine: A study in Modern Medical Missionary Development,"[1] can be considered to have achieved a satisfactory success in his endeavour to deal with each of these matters. In doing so he has produced an entirely readable and very engrossing book for those interested in modern missions, modern medicine and modern China.

The scope of the work may be indicated by a few excerpts from the contents: "The Rationale of Medical Missions," "Features of Modern Medicine, Its Demand for Exact Scientific Truth," "Chinese Medicine," "Coming of the Western Physician," "The New Profession in China," "The Advent of the Chinese Nurse," "Preventive Medicine," "The Outlook To-Day," "Policy of the Future," "Chinese Leadership."

A few quotations will serve to indicate its spirit: "It is not a strong, healthy, educated China that the world has to dread; it is an ignorant, unhealthy, impotent China that may well be a menace to civilization." "To shape the tradition of the Chinese nurse of the future and thus to inspire the whole profession with the loftiest ideals, is a task worthy of the best nurses whom the homelands can produce."

The author quotes the President of the National Medical Association of China, Dr. Wu Lien-Teh: "However necessary modern medicine may be to this country, I would rather see fewer well-qualified men and women with high ideals than a great many poorly-qualified practitioners with indifferent ambitions. The one class leads to progress; the other to stagnation, if not retrogression."

While the book is primarily intended to subserve the diffusion of Christian principles and practice, it must be conceded that Professor Balme uses his story to illustrate also a wide and scholarly conception of the world-embracing scope of modern scientific medicine.

A preface by Sir Donald MacAlister gives an initial weight and authority to the book, which acquaintance with the contents proves to be an unessential though arresting and forceful adjunct.

Some excellent illustrations, useful appendices of information, a bibliography and a very pleasing "get up" assist in making this book worth reading and rumination by medical men whose interests are not restricted within the narrower confines of the healing art.

[1] "China and Modern Medicine: A Study in Medical Missionary Development," by Harold Balme, F.R.C.S. (Eng.), D.P.H., with a Preface by Sir Donald MacAlister, K.C.B., M.D.; 1921. London: United Council for Missionary Education; Post 8vo., pp. 224. Price: 5s.; and in paper cover, 3s. 6d. net.

The Medical Journal of Australia

SATURDAY, MAY 20, 1922.

Plague Anti-Serum.

IN this issue we publish an interesting discussion on the present outbreak of plague in Sydney. Fortunately the number of infections in man has been very small. It was therefore impossible for any of the speakers to contribute anything new to our knowledge of the clinical aspect of the disease. Nevertheless, the demonstration given by Dr. R. J. Millard, C.M.G., C.B.E., was strikingly clear and has a large didactic value. Similarly Dr. Armstrong dealt very thoroughly with the epidemiology of plague. The control on the sources of infection was carried out so carefully and well that in nearly every instance it was possible to trace the infection in man to the local rat epizootic. This aspect of the subject was amplified by Dr. Eustace Ferguson in a most useful manner. The tale of the rat and its fleas was told once again and it lost nothing in the re-telling. In a discussion lasting but a short time it is impossible to parade every known fact. Perhaps the fact might have been mentioned that the rôle of the flea was first suspected in 1897 by M. Ogata on epidemiological grounds. Simonds went a stage further in 1898 by infecting rats by means of fleas that had been fed to plague rats. In 1903 Ashburton Thompson revived Ogata's hypothesis. Simond's experiments were repeated and extended by several observers between 1902 and 1904. This matter was then thoroughly investigated by the Commission for the Investigation of Plague in India and the method of transmission was definitely proven. Another point of considerable interest has been demonstrated by Professor C. J. Martin, F.R.S.. It is that *Ctenopsylla musculi*, the mouse flea, does not bite man; *Ceratophyllus fasciatus*, the brown rat flea, bites man readily and *Xenopsylla cheopis*, the Indian rat flea, bites man greedily. Dr. F. Tidswell has shown that, while *Xenopsylla cheopis* is by far the most common flea found on rats in Sydney (62%), *Ceratophyllus fasciatus* is relatively rare as compared with the non-biting *Ctenopsylla musculi*. The first named is abundant in February, March and April and becomes almost rare in the five last months of the year.

Much discussion has taken place concerning the value of serum in the treatment of plague. The significance of what has been said may be rendered more evident by a brief record of the history of the various attempts to produce an efficacious serum. In 1894 Yersin published in the *Annales de l'Institut Pasteur* a description of his method of immunizing horses with dead and living plague bacilli. A year later Calmette and Borell amplified this description. Yersin found that it was possible to inject dead bacilli in increasing quantities and to produce a protective serum. The protective power of the serum was slight at first, but the horse became immune to considerable doses of living bacilli. The immunization was therefore continued by means of virulent living plague bacilli and finally a filtrate of a broth culture was injected. The process was found to be extremely slow and the full power of the serum was not attained for a year or at times eighteen months. It was shown that this serum had practically no antitoxic action. In animals (monkeys) infected artificially, the serum exerted a curative effect if given up to twenty-four hours after infection. If given later, all the animals died. The early trials were carried out in Karachi. A distinct reduction of mortality was recorded; but this was somewhat discounted by the observation that the mortality among the patients in the period immediately following the trial was scarcely higher. Moreover, the late Sydney Rowland subsequently produced a mass of evidence which went to prove that Yersin's serum, fully matured, did not reduce the mortality among patients with plague from whose blood no plague bacilli could be grown when 0.25 cubic centimetre was used.

In 1897 Lustig and Galeotti introduced a different method of production. They cultured plague bacilli on agar-agar plates and dissolved the bacilli in 1% solution of potassium hydrate. From this solution a nucleo-protein was precipitated by the addition of acetic acid. After repeated washing, this substance was injected into horses in doses increasing

from 0.4 gramme to 1.5 grammes. In the course of three months an active antitoxic serum was produced. While the neutralizing power of this serum was supposed to be high, it is doubtful whether it could be regarded as quite satisfactory. It is true that the Indian Plague Commission reported favourably on it and Choksy claimed a considerable reduction of mortality. A little later the Bern serum was produced by an active immunization with bacilli and an immunization wth Lustig's nucleo-protein. It is thought that this serum is more active and more powerful as a therapeutic agent than Lustig's serum. Both these antitoxic sera were shown to have a relatively high protective power in laboratory animals.

In 1906 the late George Dean used filtrates from old broth cultures of plague bacilli as his toxin and obtained moderately good results. Rowland introduced a special process of extracting the nucleoprotein by means of sulphates in 1911. His experiments were conducted with much ingenuity and labour. The details cannot be reproduced in this place, but they are interesting and instructive and should be read by all who are desirous of forming a judgement on the merits of the various forms of anti-plague serum. He showed that after a course of immunizing of a little more than three months the serum was capable of neutralizing between three hundred and four hundred rat minimum lethal doses of toxin. This antitoxic power is not high, but it is distinctly higher than that of any other serum. He found that it had a more powerful protective action. Its curative power was demonstrated to be more evident than that of Yersin's serum, but it must be admitted that, even under the most favourable conditions, no benefit accrued in patients from whose blood plague bacilli could be cultured when 0.25 cubic centimetre was sown. A definite, albeit slight, reduction of mortality was obtained when the serum was given before the blood contained sufficient bacilli to permit of a culture being made from one quarter of a cubic centimetre. Later it was shown that it was quite essential to employ the same strains of bacilli in making the nucleoprotein as were involved in the particular epidemic. This means that an imported plague serum is quite useless. The evidence available at present would indicate that Rowland's serum and the Bern serum may be of value in the treatment of plague. No opinion of value can be expressed as to the efficacy of a serum unless its protective power has been tested in rats or other laboratory animals and unless it can be shown that the mortality among patients treated with it is lower than the mortality among an equal number of patients treated in other ways. The expedient of the alternate patient is almost essential. But in any case it is dangerous to arrive at conclusions until a relatively large number of observations has been carried out.

HYPERGLYCÆMIA AND DIABETES.

In the study of diabetes it is now fully recognized that the physician must not be guided merely by the presence of glycosuria. The attendant hyperglycæmia is of paramount importance. Study of this phenomenon is of interest, not merely as throwing light on the general metabolism of the body, but from a diagnostic point of view. Many authorities regard it as of considerable use in guiding and controlling treatment. The amount of blood sugar that is present in normal individuals varies from 0.07% to 0.14%. Glycosuria does not occur until the "glucose excretion threshold" has been passed. Claude Bernard originally declared that 0.03% of sugar in the blood indicated the maximum threshold and a permanent glycosuria. It has been found, however, that in many individuals the level is much lower than this. If glucose be ingested by a normal individual, a rise in the blood sugar content occurs. Absorption is rapid. The rise is constant for the given individual and generally reaches its maximum point at the end of the first hour. The level at the end of the second hour is usually lower. In patients suffering from a renal glycosuria no hyperglycæmia occurs and in fact the blood sugar curves may reveal a hypoglycyæmia three or four hours after the ingestion of a glucose test meal. The condition of renal glycosuria was dealt with in this journal in the issue of October 1, 1921.

Dr. James W. Sherrill and Dr. Henry J. John[1] have recently made a series of experiments on various individuals for the purpose of studying the influence of glucose ingestion on diuresis and blood composition in diabetic and non-diabetic persons. They record the blood sugar curves obtained in these experiments. The tests were performed in nineteen individuals, including five non-diabetic persons, nine patients with mild and five with severe diabetes. The test dose of glucose was generally one hundred grammes, but this was reduced to twenty-five, thirty or fifty grammes for some of the diabetic patients. In the normal individuals the amount of blood sugar rose rapidly and soon fell again to normal. In patients with mild diabetes the average plasma sugar during fasting was 0.126%. After glucose ingestion the composite

[1] *The Journal of Metabolic Research*, January, 1922.

blood sugar curve for the nine patients rose at the end of an hour to an average maximum of 0.26%. In patients with severe diabetes the plasma sugar for the preliminary period average 0.365%. Following glucose administration the plasma sugar percentage rose steadily and reached an average maximum of 0.545% at the end of one hour. In this series of experiments the arbitrary method of fixed dosage was used with a few exceptions.

Dr. W. H. Olmsted and Dr. L. P. Gay[1] have recently made a very complete study of the blood curves in more than two hundred patients who had been given a standardized glucose meal. These authors state very definitely that the results of blood sugar determinations will be made more constant if the weight of the patient on whom the experiment is to be made, be taken into consideration in fixing the dose of glucose to be ingested. They state that it is obvious that the same weight of glucose should not be given to patients of different weights. It is held that blood volumes varies approximately with weight. In health sugar is stored both in the liver and the muscles and it is possible that additional sugar storage might take place, in some abnormal conditions, in other tissues. These authors claim, therefore, that the weight of the individual is the best index to his available space for storing glucose. The method that the authors have used in determining the dosage may be summarized as follows: The percentage of injected glucose absorbed in two hours has been shown to be from 66 to 80. The maximum intravenous tolerance rate of glucose for man and animals without glycosuria has been shown to be 0.85 grammes per hour per kilogram of weight. Taking these factors, the dose would be $(2 \times 0.85 \times \frac{100}{66})$ or about 2.5 grammes of sugar per kilogram. They, however, accept Janney's recommendation of 1.75 grammes per kilogram of weight. This amount of glucose for a man weighing 68.5 kilograms, for instance, would amount to 120 grammes of glucose or 480 calories. This dose is greatly in excess of caloric needs under ordinary circumstances and would show the ability of the individual to store the excess of an amount of sugar commensurate with his weight absorbed in a unit of time. The patients tested by Drs. Olmsted and Gay included some who were suffering from other conditions, as well as some suffering from diabetes. In the diabetic patients the most typical results were obtained in patients suffering from a mild form of the disease. In the case of these patients the hyperglycæmia was sustained to a more marked degree than in any other condition affecting glycogenesis. At the end of three hours the hyperglycæmia was commonly above 0.3%. It is in cases of mild diabetes that the diagnostic value of blood sugar curves after a standardized glucose meal is most pronounced.

The estimation of sugar present in the blood of a given patient suffering from diabetes may be helpful from the point of view of treatment. It has very often been found that, after a patient has been under treatment for some time and his blood sugar percentage has fallen below his sugar threshold, that the threshold tends to rise. If the treatment is ordered on evidence obtained from the urine, the patient will be treated differently than if the treatment aims at the maintenance of a normal blood sugar level. This is the correct standard of treatment.

[1] *Archives of Internal Medicine*, March 15, 1922.

THE TRANSMISSION OF MEASLES TO GUINEA-PIGS.

DURING the past five or six years a considerable amount of work has been conducted with the view to disclose the nature and characters of the virus of morbilli. In 1905 Hektoen demonstrated that, notwithstanding the failure of all attempts to isolate and identify this virus, the blood of persons suffering from the disease could convey the infection to monkeys. Goldberger and Anderson proved that the infectivity was not lost when the infected blood was passed through a close porcelain filter. In 1919 Sellards and Wentworth published experiments to show that monkeys could not be infected with the blood of patients suffering from measles. Notwithstanding the evidence of their experiments, the majority of bacteriologists still regard the work of Hektoen as sound. Drs. Charles W. Duval and Rigney D'Aunoy have now shown that guinea-pigs can be infected with the blood of persons taken during the acute stages of morbilli.[1] When blood taken during the stage before the appearance of the rash and at a period twenty-four hours after the defervescence was used, no reaction was produced in the guinea-pig. One or two cubic centimetres of defibrinated blood were injected into the heart of the guinea-pigs within thirty minutes of the bleeding. The effect comprised a pyrexia of one or two degrees centigrade, appearing on the ninth day and lasting for three or four days, accompanied by a distinct leucopenia which lasted about eight days. The guinea-pigs remained apparently well during the pre-febrile period, but became listless, depressed and ill during the pyrexia. The leucocyte count fell from 12,000 to as low as 3,600 per cubic millimetre. Intra-cardiac injections of blood of normal persons did not produce fever or leucopenia and did not disturb the general health of the guinea-pigs. Some of the animals died during the attack. These animals and others killed at the height of the reaction were examined *post mortem*. An acute hæmorrhagic nephritis was found in every instance. At times the changes were very extensive. A further series of experiments was conducted with the blood of guinea-pigs infected with the blood of measles patients. The authors were able to produce the same syndrome in fresh guinea-pigs by repeated passage of the virus. In no instance was any rash seen, but a slight coryza occurred in a few of the infected guinea-pigs. These experiments, if confirmed, would prove that the measles virus is pathogenic for guinea-pigs and that the infection can be conveyed to them in the blood of persons suffering from measles during the acute stage. Apart from the possibility of discovering the virus in experiments along these lines, this work might be of value in the diagnosis of atypical morbillar infections.

[1] *The Journal of Experimental Medicine*, February 1, 1922.

Abstracts from Current Medical Literature.

SURGERY.

Gastric Crises of Tabes Dorsalis and Their Surgical Treatment.

R. C. SHAW (*British Journal of Surgery*, January, 1922) deals with the gastric crises of *tabes dorsalis* and their surgical treatment. The first half of the article is devoted to the discussion of the nervous paths involved in the production of the pain of gastric crises. The pain is of a composite character, being of two forms. The first is felt superficially in the body wall and is of two types. One is sudden and shooting and localized along the dorsal nerves, the other being dull and constricting and not well localized. The second form of pain in a crisis is felt in the epigastric region, generally to the left of the mid-line. It is severe and griping and accompanied by deep tenderness. After section of the posterior nerve roots, from the fourth to the eighth dorsal nerves inclusive, the pain which shoots along the course of the dorsal nerves is relieved, but the dull, constricting pain persists. In one patient who had posterior rhizotomy performed, the shooting and constricting pains were both relieved; but this patient had atrophy of his intercostal muscles. In two patients upon whom a similar operation was done and who did not have intercostal atrophy the dull, constricting pain persisted, as did also deep tenderness in the area supplied by the divided nerves, although cutaneous anæsthesia was present. It is suggested from this that some of the afferent sensory fibres pass by way of the anterior roots. In two cases reported by Head the posterior cervical roots of the fifth, sixth and seventh segments were divided, but still deep sensibility remained in the area of cutaneous anæsthesia. The second type of pain, localized in the left of the epigastrium, may be termed visceral. Severe and griping in character, it still persisted after the root section. It is probably due to stimulation of afferent nerves in the stomach wall. These are fibres contained in the vagus nerve and sympathetic nervous plexus. The irritation of the motor vagal fibres produces the vomiting of the crisis. This was not relieved by the section of the posterior roots. Exnar in one of his patients divided the vagus and completely relieved the deep griping pain, but in others, in spite of vagotomy, the pain persisted. Stimulation of the sympathetic in one of the cases of posterior rhizotomy was followed by a typical gastric crisis, so that he concludes that both the vagi and sympathetic nerves conduct the visceral sensory impulses, the sympathetic being probably the more important path. The vomiting of the crisis is produced by irritation of vagal or sympathetic afferent fibres which react on the motor nucleus of the vagus. In the operative treatment of the condition complete recovery seems dependent upon the interruption of afferent sympathetic impulses. This may be produced by dividing the antero-lateral ascending tract of the spinal cord between the second and third dorsal nerves, as done by Souttar, or by division of the posterior roots in the thoracic region. This latter reduces to a minimum the afferent impulses going to stimulate the vagal motor nucleus. Division of the vagus has important physiological sequelæ, the chief of which is gastric stasis. Probably the most satisfactory results follow posterior rhizotomy.

Eventration of the Diaphragm.

E. H. FUNK AND W. F. MANGES (*American Journal of the Medical Sciences*, September, 1921) discusses eventration of the diaphragm and reports a case. This rare condition was described by Petit in 1790. If consists of a thinning and relaxation of the diaphragm, with marked elevation. They consider the condition to be congenital, as it is found in the new born, is often associated with congenital abnormalities and involves the left side of the diaphragm, which is so complex in its development. Their patient had pain in the epigastrium and left side of the chest. Examination showed a well nourished patient with a well formed and symmetrical chest. Percussion gave a tympanitic note at the third rib in the anterior axillary line, merging in the tympany of the stomach below. No coin sound was present. A provisional diagnosis of hydro-pneumothorax was made, but paracentesis in the left sixth interspace of the mid-axillary line was negative. X-ray examination showed that the left half of the diaphragm was raised and dome shaped and did not move on forced inspiration. The diagnosis is made in most cases by the X-ray, but it is pointed out that inspection of the movements of the costal margin, as suggested by Hoover, is of great assistance. On inspiration there is widening of the subcostal angle, owing to divergence of the left costal border. The high position of the diaphragm places it at a mechanical disadvantage in its function as an antagonist to the intercostal muscles.

Carcinoma of the Bladder with Bone Metastases.

HERMAN L. KRETSCHNER (*Surgery, Gynecology and Obstetrics*, February, 1922) discusses carcinoma of the bladder with bone metastases. He thinks that secondary growths in bones are much commoner in carcinoma of the bladder than the records show. Further, these secondary growths occur much earlier in the course of the disease than is described. Malignant growths in certain regions, such as the breast, prostate, thyreoid gland and supra-renal glands, have been known for a long time to be specially liable to produce secondary growths in bones. To this list Kretschner thinks the bladder should be added. He describes three cases of his own. In each instance pain was present. It was of two varieties. In one it was due to the bladder growth and in the other to the bone involvement. In this latter the pain was excruciating, requiring much morphine for its relief. It was situated in the region of the involved bones, *viz.*, lower spine, pelvic girdle and thighs. Cystoscopy showed that, although bone metastases had occurred, the primary growth in the bladder was small. Skiagrams showed multiple foci and massive bone destruction, without any pronounced sign of new bone formation. The bone involvement was therefore of the osteoclastic type, unlike the osteoplastic type marked by much new bone formation seen in secondary bone growths following carcinoma of the prostate.

The Choice of Operation in Inguinal Hernia.

P. G. SKILLERN, JUNIOR (*Surgery, Gynecology and Obstetrics*, February, 1922) writes on the choice of operation in inguinal hernia. He divides the various types of hernia into two main groups. In the first group the sac is stalked. This embraces hernia in infants and young adults, in whom the sac is long, narrow and pedunculated, being in fact the *processus vaginalis* or the funicular process. In such patients the muscular valve of the inguinal canal is efficient and all that is necessary is high ligature of the sac. In the second group, that is, those with sessile sacs, something more is required than simple ligature of the sac. The sessile group includes the variety known as "diastasis of the external oblique aponeurosis." In this class an oval bulging, generally bilateral, is present just to the outer side of the external (subcutaneous) inguinal ring. Palpation shows a deficiency in the wall of the inguinal canal in this region, the edges of the opening being easily felt. There is no true hernial sac, but a loose peritoneal pouch forced through the opening. It is not necessary to open or ligate the sac, but merely to reconstruct the weakened wall of the canal. The "diastasis" is akin to the condition called "direct" hernia, in which the hernia protrudes to the medial side of the internal ring. In this class the conjoined tendon (*falx aponeurotica*) is thinned out and offers but a weak barrier to the hernia: Sometimes the peritoneal protrusion overflows to the outer side of the inferior epigastric artery and a hernia called the "pantaloon" type appears. In the treatment of direct hernia Skillern frees the cord from the sac right up to the internal ring, ties off the sac and sutures with interrupted stitches the lower edge of the internal oblique muscle to the grooved surfaces of Poupart's ligament. The edge of the lower flap of the external oblique muscle is then stitched to the anterior surface of the internal oblique muscle and the conjoined tendon and, lastly, the edge of the upper flap of the external oblique muscle is brought in front of the lower flap. The cord is brought out in front of the external oblique muscle.

GYNÆCOLOGY AND OBSTETRICS.

Ectopic Pregnancies.

JOHN OSBORN POLAK (*American Journal of Obstetrics and Gynecology*, September, 1921) has made a study of 307 cases of ectopic pregnancies. Only eight persisted beyond the twelfth week. Of these, three were continued to full term within the abdomen and five terminated in the secondary rupture of an intra-ligamentous pregnancy at the third, fourth and fifth months respectively. There were three groups: (*i.*) There were 186 women who had had a previous infection following marriage, intra-uterine instrumentation, abortion or child birth or peritonitis. (*ii.*) This group comprised 94 women with a history of dysmenorrhœa with developmental defects, such as funnel pelvis, infantile uterus, narrow vagina; the women had remained sterile after marriage for varying periods and finally, after some measures had been adopted for the cure of the sterility, an ectopic gestation followed. (*iii.*) The last group included 27 women who had had previous normal pregnancies. The sac was found to be interstitial in six, in the isthmus in 79, in the ampulla and free portion in 203, in the stump of a previously amputated tube in three and in an angulation of the tube caused by a previous operation for internal shortening in eight. Blood was noted in the abdominal cavity in all the patients, whether the sac had or had not ruptured. Free blood in varying amount was usually present. Infected hæmatoceles occurred in three patients, three died before operation could be performed and 296 were operated on, of whom seven died (one on the table from hæmorrhage and five from peritonitis). The history is of the most important diagnostic value. The decidual reaction in the tube produces a decidua not thick enough to protect the underlying muscle and venous radicals from the erosive action of the syncytial cells, which, by riddling the vessels, causes hæmorrhage into the decidua. This tends to unseat the ovum, over-distends the tube and causes pain and bleeding, the pain being of a colicky type. As the tubes and uterus have a common origin, this unrest or peristaltic wave is transmitted to the uterus and leads to uterine contractions, with slight bleeding from the endometrium. A definite history of anomalous uterine bleeding was obtained from 227 women. This appeared either as prolonged menstruation or as a metrorrhagia after a varying period of amenorrhœa. There is also a slight leaking of blood through the abdominal ostium into the peritoneal cavity. The peritoneal reaction explains the occurrence of four symptoms: (*i.*) A slight elevation of temperature, which occurred in 80% of all the patients under observation for several days prior to the making of the final diagnosis. (*ii.*) A moderate leucocytosis. (*iii.*) Exquisite sensitiveness of the cervix to motion, which was present in all the women subjected to physical examination. (*iv.*) Pain on defæcation as the hard fæcal mass passes between the sensitive utero-sacral ligaments. This is epecially noticeable in left-sided tubal pregnancies. The uterus is enlarged because it contains a decidua which has been prepared in anticipation of the reception of the ovum. The cervix is soft, as a result of congestion; but this symptom is variable and of no diagnostic value. On bimanual examination the practitioner does not recognize the usual diagnostic sign of pregnancy in the uterus, *i.e.*, the elasticity of the median portion of its anterior wall and the compressibility of its isthmus. The uterus is displaced because the tubal tumour, owing to its weight, has fallen into the lateral, the posterior or the anterior *cul de sac*. The tumour displacing the uterus has certain definite characteristics. It is of rapid growth, exquisitely sensitive, because the tubal covering is stretched to its utmost, and it is fluctuant. When rupture or abortion occurs, there is sudden pain, with shock of greater or lesser degree. If hæmorrhage is slight, the patient will react, but if severe she collapses. If the condition is diagnosed the tube is removed. If rupture occurs, with severe shock and hæmorrhage, Polak advises rest and morphine until the patient has reacted. No saline solution is injected nor blood transfused until after operation.

Ovarian Extract in Menstrual Disorders.

JOHN COOKE HIRST (*New York Medical Journal*, October 5, 1921) formulates the following conclusions on the comparative value of whole ovarian extract, *corpus luteum* extract and ovarian residue in menstrual disorders. Results are not invariable with any extract, but if it be intelligently used, considerable success may be expected, except in the case of ovarian residue, which yields decidedly nebulous results. The effects are often slow to appear, particularly in menstrual disorders, and patience is one of the prime requisites in both patient and doctor. Results are promptest in the menopause with whole ovarian extract and in the nausea of pregnancy with *corpus luteum* extract. Co-operation between physician and patient is needed and over-optimistic statements are to be avoided. The results may be slow in appearing and may leave much to be desired. The most discouraging results are met with in the cases of obesity and amenorrhœa.

Torsion of Uterine Adnexa.

R. R. SMITH AND W. J. BUTLER (*American Journal of Obstetrics and Gynecology*, November, 1921), in reporting a case of torsion and strangulation of a normal right uterine tube and ovary in a child aged nine years, reviewed the literature since 1900 on this subject and arrived at the following conclusions: Torsion of ovarian tumours is an uncommon occurrence in childhood, as shown by the fact that only twenty-six cases, including their own, have been reported in the literature since 1900. Of these, about 50% occurred between the ages of eight and ten; 60% of them were dermoids. Tumours varied in size from that of an adult ovary to one reaching above the umbilicus. The symptoms are those of an abdominal crisis similar to that of the same accident in adult life, *viz.*, sudden abdominal pain, vomiting, a variable degree of sweating, tenderness, rigidity, distension, fever, increased pulse-rate and the presence of an abdominal tumour. The diagnosis is usually difficult, probably owing to the rarity of the condition. Appendicitis is often the pre-operative diagnosis. The causes are much the same as in adult life. Torsion of normal adnexa has been reported to date as occurring in fourteen patients. Of these, eight occurred before the age of twenty; four under twelve and presumably before puberty. The tube alone may be involved, the tube and ovary together or the ovary may be essentially the offending organ. A large percentage occur in relation to the menstrual period. Three are reported as occurring during pregnancy. Some question still exists as to whether a normal tube can undergo torsion, but it seems to be proved that a normal ovary may do so. Factors to be considered are the length of the mesentery, the size of the ovary, the length of the tube and the histological structure of the organ. Torsion of adnexa in hernial sacs (apart from strangulation) is relatively rare. It occurs only in congenital inguinal hernias and usually in the first year of life.

Experimental Studies Following Öophorectomy.

HAROLD BAILEY (*American Journal of Obstetrics and Gynecology*, July, 1921), reviewing the literature on metabolic studies after öophorectomy, implantation experiments and the evidences of relationship between the uterus and ovaries, states that the metabolic studies tend to show a reduction both of the oxygen consumed and the carbon dioxide eliminated. The greatest reduction occurs some two or three months after the operation and when atrophic changes have probably occurred in the uterus. The experimental work must be considered inconclusive in regard to the demonstration of a direct effect on the energy metabolism of the cell by an ovarian secretion. Experiments to ascertain the condition of the ovary after hysterectomy show that in young animals the ovarian development goes on in a normal manner. Work with adult animals would indicate that, following hysterectomy, no ovarian change may be demonstrated until after a three- or four-month period. Clinical gynæcology offers proof that the menopause ensues notwithstanding that the ovaries remain, if the uterus is entirely removed, and that saving a portion of the uterine mucous membrane, so that menstruation may occur, prevents these symptoms. Transplantation of the ovary is of no value in relieving menopausal symptoms unless the uterus or part of it remains.

British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE WESTERN AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on March 26, 1922, at the Hospital for the Insane, Claremont, DR. G. W. BARBER, C.B., C.M.G., D.S.O., the President, in the chair.

Financial Statement.

DR. W. TRETHOWAN, the Honorary Treasurer, read his report and presented the balance sheet for the year ended March 26, 1922. He moved the adoption of the financial statement and its incorporation in the minutes.

The motion was seconded by DR. J. J. HOLLAND and carried.

DR. TRETHOWAN reminded members that if the Australasian Medical Congress (British Medical Association) held its Second Session in Perth in 1926, the funds of the Branch could not be used for this purpose. A special subscription would have to be made by the members.

Report of the Council.

THE PRESIDENT then proceeded to read the Annual Report of the Council:

REPORT OF THE COUNCIL FOR THE YEAR 1921-1922.

At the beginning of this year the membership amounted to one hundred and fifty-one, being three less than last year. The decrease is due to more members being transferred out of the Branch to other States than into the Branch. The total number of practitioners in the State is one hundred and ninety, so that 79% of these are members of the Western Australian Branch of the British Medical Association, which is an excellent proportion; but I trust all members will endeavour to induce the remainder to join our Branch, which is an undoubted benefit to all practitioners.

The Council has held eleven meetings, with an average of six attendances. There have been eleven general meetings, with an average of twenty-six attendances. The Council and the general meetings have considered all important items brought before them, the principal items being the Model Lodge Agreement and the *Hospital Bill*.

London Representation.—DR. T. P. DUNHILL was appointed to represent this Branch on the Council in London.

MR. HADLEY represented this Branch at the Annual General Meeting held at Newcastle-on-Tyne, England. Mr. Hadley's report is reproduced below. The Council has to thank him for the good work he did.

DR. C. W. T. WOODS has been asked to represent this Branch at the coming Annual Meeting, to be held at Glasgow in July.

Annual Subscription.—The general meeting last September reduced the annual subscription to country members from £4s. 4s. to £3 3s..

During the year one meeting of the Ethical Committee was held with satisfactory results.

General Meetings and Papers.—Most of the meetings were held at the Perth Hospital, but through the kindness of DR. J. T. ANDERSON, DR. BASSETT and DR. MCGREGOR, one meeting was held at the Hospital for Insane, Claremont, one meeting at the Base Hospital and one meeting at the Children's Hospital. During the year interesting papers were read by DR. BENTLEY, DR. MOSS, DR. JUETT, DR. MCWHAE, DR. MORLET, DR. D. SMITH, DR. MCGREGOR and DR. HOLLAND for DR. BEVERIDGE.

The annual dinner was held at the Savoy Hotel in December and was well attended, but the alteration to a later date than usual did not result in any increased attendance of country members, as was hoped.

Mr. F. A. Hadley's Report on the Annual Meeting at Newcastle.

The Annual Meeting of the British Medical Association at Newcastle-on-Tyne was preceded by a conference in London, at which Dr. Todd, supported by representatives from the Australian Branches, most ably proposed that there should be made an alteration in the regulations which would allow Australia to manage her own affairs and finances without having to refer all such matters to the Council in London as at present. Much discussion followed, with some opposition, chiefly on the grounds that the present Articles would not permit the change, legal opinion being divided on the subject. Canadian and South African representatives strongly supported the motion, which was carried and referred to the Representative Body at Newcastle, which confirmed it.

Much interesting discussion took place in this body, which is practically a medical parliament.

It is true nearly all the subjects dealt with matters in the British Isles, but many of them, especially in connexion with hospitals and the *Insurance Act*, may at any time in the near future occur in Australia. The high tone taken in the proceedings was a lesson in itself.

The town of Newcastle laid itself out to insure that visiting members should have a pleasant as well as an instructive week.

Presidential Address.

THE PRESIDENT then read his address (see page 539).

Election of Office-Bearers.

THE PRESIDENT then announced the result of the election of office-bearers and members of the Council for the ensuing year:

President: DR. D. D. PATON.

Vice-President: DR. D. M. MCWHAE, C.M.G., C.B.E..

Honorary Secretary: DR. T. L. ANDERSON.

Honorary Treasurer: DR. W. TRETHOWAN.

Members of Council: DR. A. H. GIBSON, DR. R. C. E. ATKINSON, DR. E. A. OFFICER.

Members of the Ethical Committee: DR. H. B. GILL, DR. W. N. NELSON, DR. W. P. SEED.

DR. TRETHOWAN pointed out that twelve members had been nominated for election to the Council, but the nominations had not been seconded. He suggested that in future members should take steps to insure that all nominations were by two members.

Induction of President.

DR. BARBER then invited DR. D. D. PATON to take the chair. He said that the choice of the members had been very fortunate, as he considered Dr. Paton most suitable to pilot the Branch through an important year. The negotiations in connexion with the *Hospital Bill* and the finalizing of the agreements with the friendly societies and timber mill societies would occupy their attention during the coming year. He wished to thank the Council and particularly the Honorary Secretary for the support extended to him and for their untiring efforts in dealing with the large amount of work undertaken during the year.

DR. D. D. PATON then took the chair.

Election of Honorary Auditors.

DR. M. K. MOSS and DR. A. E. RANDELL were elected Honorary Auditors for the ensuing year.

Dementia Præcox.

DR. J. T. ANDERSON read a paper entitled "Dementia Præcox" (see page 540).

DR. J. BENTLEY, M.C., exhibited patients suffering from hebephrenia, catatonic stupor and catatonic excitement, *dementia paranoides*, neologism, verbigeration and mannerisms and others to demonstrate the effect of ductless gland therapy.

DR. D. M. MCWHAE, C.M.G., C.B.E., thanked Dr. Anderson for his paper and Dr. Bentley for his demonstration of patients. He considered that the paper would be a valuable guide to medical practitioners who were called upon to deal with conditions of this kind in private practice. He was of opinion that the treatment with ductless glands was full of great possibilities.

Annual Meeting of the British Medical Association.

THE HONORARY SECRETARY read a letter from SIR WILLIAM MACEWEN, C.B., President-Elect, inviting members of the

Branch to attend the Annual Meeting of the British Medical Association, to be held in Glasgow in July, 1922.

Expression of Thanks.

DR. PATON proposed a vote of thanks to Dr. J. T. Anderson for his kind hospitality to members. The motion was carried by acclamation.

In his reply DR. ANDERSON stated that he was always pleased to have a meeting of the Branch at the Hospital, not only to meet the members, but also to be able to bring before them patients they would otherwise not be able to see.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, on April 7, 1922, DR. T. W. LIPSCOMB, the President, in the chair.

Plague.

DR. W. G. ARMSTRONG read a paper on the outbreak of plague in Sydney (see page 546).

DR. R. J. MILLARD, C.M.G., C.B.E., gave an account of the clinical aspects of the disease in the patients treated at the Coast Hospital. He stated that he had had the opportunity of observing and treating thirteen patients out of the total of sixteen persons infected in Sydney. There were two points of interest with which he would deal. The first was the diagnosis and the second was the effect of specific treatment. Dr. Armstrong had dealt exhaustively with the symptoms, clinical course and physical signs. There was therefore not much left for him to say concerning the diagnosis. The diagnosis was neither elaborate nor difficult. It rested on the presence of a bubo which was distinguishable from a bubo of venereal disease and from septic adenitis. The gland was tender and was surrounded by an area of œdematous and inflamed tissue. It could usually be examined with very little difficulty. In order to confirm the suspected diagnosis of plague a bacteriological examination of the contents of the gland had to be made. A syringe was required for this purpose, armed with a needle of moderate calibre. The needle was introduced into the bubo and the piston worked backwards and forwards several times until a minute quantity of the material was sucked into the syringe. This was at once expressed on to an agar culture tube. A smear was made immediately and at the same time the culture tube was incubated. Often the smear was found to contain large numbers of plague bacilli. In the event of no typical plague bacilli being recognized in the immediate smear, it was necessary to wait for the development of the bacilli in the culture tube, perhaps two or three days.

The onset was usually sudden. The second patient of the series, a man aged sixty years, was on the rifle range on Thursday, December 1, 1921, in the afternoon. When he arrived home in the evening he was acutely ill, had pain in the groin and vomited. A femoral bubo developed almost immediately. He was admitted to the Coast Hospital on December 3, when there was a typical bubo with peri-adenitis. Dr. Millard pointed out that the bubo was usually in the groin. The lower limbs presented a greater area for fleas and consequently the bites were usually situated in this area. In one patient the bubo was in the axilla. It was stated that the prognosis of plague with cervical or axillary bubo was more serious as a rule than when the bubo was in the inguino-femoral region. The patient with the axillary bubo was a girl who had been taken ill on January 25. Her temperature was 39.1° C.. On the following day the axillary glands were tender. She became delirious during the afternoon. On January 27 the temperature was normal, but on January 28 the fever returned. In the evening the temperature was 40° C. when she was admitted to the Coast Hospital. There was œdema and infiltration in the axilla. A small phlyctenule was discovered about the level of the nipple. Dr. Millard regarded this as the probable site of the infecting flea bite. *Bacillus pestis* was obtained in a culture made from this phlyctenule. Another phlycteular sore was found in the groin and plague bacilli were obtained from this as well.

Dr. Millard proceeded to exhibit on the screen the temperature charts, with full annotations of each patient admitted.

The second patient of the series had been taken ill on December 1 and admitted on December 3. On admission he received an intra-muscular injection of fifty cubic centimetres of Yersin's anti-plague serum. The dose was repeated on the second and again on the third day. The temperature fell from 39° C. to 37° C. (102.2° F. to 98.6° F.). Later the bubo was incised and for a short time there was slight fever. The necrosed gland was enucleated on the nineteenth day. Dr. Millard referred to a description by Dr. E. H. Binney in THE MEDICAL JOURNAL OF AUSTRALIA of November 5, 1921, page 399, of the manner in which a necrosed gland popped out of the area of peri-adenoid suppuration like a kernel out of a nut. The only other point of interest in regard to this patient was the occurrence of albuminuria during the febrile period.

The third patient was admitted on January 28, 1922, on the third day of illness. This was the girl with the axillary bubo. On admission the temperature was 39.1° C. (102.4° F.). It rose to 40.5° C. (105° F.) on the same day. A dose of fifty cubic centimetres of serum was given on admission and again on the following evening. The temperature fell on the third day and did not rise again. The bubo was incised on the fifteenth day.

The fourth patient was taken ill on February 15, 1922, with a left inguino-femoral bubo. He was admitted to hospital on February 18 with a temperature of 40.5° C. (105° F.). An injection of one hundred cubic centimetres of serum was followed by a drop of temperature with a subsequent rise. On the following day a further dose of one hundred cubic centimetres was given. The temperature continued to fall. On February 20 fifty cubic centimetres were injected and on February 21 the temperature reached normal. It rose again on March 1 and it was discovered that an abscess had formed at the site of injection. Dr. Millard pointed out that this patient had been in a very dirty condition on admission and that the illness was so desparate that the injection of serum was given before the nurses had had an opportunity to cleanse his skin. Subsequently the patient had an attack of pneumonia with pleurisy, which was associated with high fever lasting for several days. During convalescence from pneumonia œdema and pain of the left calf, ankle and foot appeared. The œdema disappeared after the bubo had been incised and the necrosed gland had been enucleated. The patient ultimately recovered.

The fifth patient had been taken ill on February 17 and had been admitted to the Coast Hospital on February 18. His temperature on admission was 39.8° C. (103.6° F.). There was a right femoral bubo. The patient received one hundred cubic centimetres of serum. On the following day the fever abated, but returned after a short time and then disappeared. Serum sickness occurred eighteen days after the injection. There were profuse urticaria and severe joint pains. The patient was treated with morphine and the usual palliative measures. Recovery was complete.

In the sixth patient the clinical manifestations started on March 9, 1922. When admitted on March 11 the temperature was 39.4° C. (102.8° F.). He was given one hundred cubic centimetres of serum. The temperature fell within the following twenty-four hours and the signs of illness gradually disappeared. Serum sickness developed eight days later. This patient had had a slight albuminuria at the time of admission which cleared up with the disappearance of his symptoms.

The seventh patient had been taken ill on March 10 and had been admitted on March 11. Plague bacilli had not been isolated, but the clinical signs of the disease were typical and there was no doubt concerning the diagnosis. There was a left inguinal bubo. The onset had been quite sudden and the patient was very acutely ill at the time of admission. His temperature was 40.5° C. (105° F.). He was given one hundred cubic centimetres of serum. Twenty-four hours later the temperature had fallen considerably and convalescence soon began. A serum rash developed six days after the injection.

The eighth patient had been taken ill with an inguino-femoral bubo on March 27, 1922. He was admitted on the same day. The temperature on admission was 39.4° C. (103° F.). An injection of fifty cubic centimetres of serum was

given. On the following day the temperature had risen to 40.5° C. (105° F.). The patient was extremely ill. A further injection of fifty cubic centimetres of serum was given. This was followed by a fall of temperature. On March 30 the temperature was again very high, having reached 40.1° C. (104.2° F.). A third dose of fifty cubic centimetres of serum was injected. The patient's condition soon improved and the temperature fell to normal. On April 5 a serum rash appeared.

The onset of the illness of the ninth patient dated back to March 29, 1922. The patient was admitted on the following day, when a left inguinal bubo was well developed. The temperature was 40.5° C. (105° F.). An injection of firty cubic centimetres of serum was given, which was followed by a fall of temperature. On April 1 the temperature was still high and a further injection of serum was administered. Recovery took place. The serum rash appeared on April 7.

The tenth patient was taken ill on March 31 and was admitted to hospital on the following day, when the temperature was 38° C. (100.4° F.). An injection of one hundred cubic centimetres of serum was given in the evening, the temperature being at that time 39.3° C.. On the following day it was thought advisable to give a further fifty cubic centimetres. The temperature began to fall soon after and reached the normal line within two days. A serum rash appeared on April 7.

The eleventh patient had been admitted on April 3, on the second day of illness. The temperature was 39.6° C. (103.2° F.). The patient looked very ill, was flushed and drowsy and had a typical right inguinal bubo. The diagnosis of plague, however, had not been confirmed bacteriologically. An injection of fifty cubic centimetres of serum was given shortly after admission. The temperature fell during the following twenty-four hours and the general condition of the patient soon improved. Dr. Millard pointed out that an attempt would be made to obtain bacteriological confirmation of a diagnosis from an examination of the bubo.

Another patient, actually the first to be admitted, had also received plague serum on admission. He had been sent in on account of signs of a prostatic abscess. This man had been employed at a produce store and had been doing hard, manual work. The onset had taken place on November 26, when the patient had suddenly collapsed. On November 28 a smear had been prepared from the puncture fluid of the bubo. This smear contained an enormous number of the bacilli. On December 1 the patient was tremulous and cyanosed. The temperature, which had been high, started to come down, but there was no improvement in the patient's condition. He became very collapsed and delirious. This patient died.

In another patient the infection was so mild that no serum was given. This patient on admission had a large bubo at the point of breaking.

One other patient had received serum, but had died on the evening of the day of admission.

DR. E. W. FERGUSON read a paper entitled "The Plague Epidemic" (see page 552).

DR. BURTON BRADLEY stated that the announcement appearing on the business paper that he would read a formal contribution on serum treatment and prophylaxis of plague had been made under a misapprehension. He proposed to discuss certain aspects of plague prevention, especially in relation to the preparation of a serum and the examination of rats. He presumed that the chief interest of the members would be centred around the serum used in the present outbreak. He pointed out that his own opinion as to the value of the serum was conservative. All he could say was that in the patients treated a strong *a priori* case seemed to have been established. When the news of plague in Brisbane came to Sydney, the idea of making a serum occurred to Mr. John Stewart, of the Biological Institute of Australasia, and to him. Cultures from a rat taken on board the *Wyreema* had been obtained through the courtesy of Dr. Armstrong. With the practical help of Dr. Harold Norrie the work had soon been in progress. The health of the horses had been safeguarded by Mr. R. Stewart, veterinary surgeon. He had discussed with several people, including those most competent to express an opinion, which method of immunization should be employed. After due consideration he had selected Yersin's method and had carried it out with some modifications on two horses. Dr. Bradley claimed that the evidence brought forward in favour of Rowland's serum as against Yersin's, in so far as its practical value was concerned, was not convincing. At first killed cultures had been injected. At a later date, when the horses were tolerating large doses, living cultures had been given. He started by giving ten milligrammes of bacilli subcutaneously and increasing the dose after a week's interval. Later on intravenous injections had been used until two hundred milligrammes of living plague bacillary substance had been injected at one time. He had used four different cultures, all grown from highly virulent local strains. The serum was first used in an immature condition, three and a half months after the immunization had been begun. This course had been adopted because it was held that it might be of some use in the absence of anything better. The agglutinating property of the mature serum had been tested and agglutination had been obtained in a titre of one in 6,000. He admitted that agglutination of plague bacilli was unsatisfactory and was difficult to determine. He had asked independent observers to check the results obtained.

Dr. Bradley referred to Dr. Armstrong's statement that the prevention of plague depended on the watching of the rat. In Sydney the method of diagnosis employed for the detection of plague in rats by experts was rapid and reliable. A very large number of animals was dealt with. During the time when he had been in charge of the Queensland Micro-Biological Laboratory, the methods adopted were quite different. Smear preparations were made in various parts of the State, often by laymen, and the slides were stained by fuchsin and not by Gram's method. Apart from the fact that the smears were frequently contaminated, this staining method was not a good one. He had pointed out on more than one occasion to the Minister that the system, which depended largely on technical work carried out by laymen, would eventually lead to an epizootic remaining undetected for three or four weeks. It was well known that the epizootic had existed in Queensland for a considerable time before it was recognized.

In dealing with the use of anti-plague serum, Dr. Bradley stated that the serum had been produced by a private firm. Mr. Stewart, the chairman, had offered the serum to the Queensland Government and to the New South Wales Government under the same conditions of free trial in each case. The New South Wales Government had accepted the offer and had used the serum. According to the reports published in the Queensland press, the Queensland Government had refused to use the serum because the Commonwealth Minister had advised that "it might be unwise." It was well known that he had been in charge of the work of preparation and he therefore regarded this expression of opinion as a personal slight and as a reflection on his professional ability. He held that Ministers of Governments were not justified in interfering deliberately with the use of something that might do good. He claimed that this action on the part of a Minister revealed the methods adopted for protecting the products of the Commonwealth Serum Laboratories. He repeated that he had no proof that the serum exerted a curative action; it remained to be shown whether any plague serum was of value.

DR. J. S. PURDY, D.S.O., stated that he had been very interested in Dr. Armstrong's paper. He was glad that Dr. Armstrong had mentioned the presumptive evidence of a second or re-introduction of plague by the *Levuka*. He dealt briefly with the facts as detailed by Dr. Armstrong which tended to support this suggestion. Before leaving this aspect of the subject, he expressed gratification at the fact that the relations between the Federal and the State authorities in New South Wales were friendly and that there was active cooperation in the attempt to deal with the outbreak.

Dr. Purdy proceeded to exhibit a series of lantern slides illustrating the various methods of rat extermination and plague control. Many of the pictures were illustrative of Egyptian methods. The first represented a patient ill of plague, to illustrate the typical facies, to which, Dr. Purdy stated, Dr. Billeli, the Director of Quarantine at Port Said, who had an extensive acquaintance with plague, had never

failed to direct his attention; the diagnosis in each case had afterwards been confirmed.

Dr. Purdy had referred to Dr. Billeli at Port Said a patient whose case his German colleague, Dr. Vay, of Suez, had regarded as not plague. Although the patient had a temperature of 39° C., rigors, headache and vomiting, together with a femoral bubo, Dr. Billeli did not consider it plague, owing to the absence of the typical facies. When the patient was removed to the lazarette the condition was proved on removal of portion of a gland to be one of tuberculous adenitis, a rare occurrence in the femoral region.

Dr. Purdy also referred to cases of tropical or firemen's buboes, which had been difficult to diagnose. He also reminded those who had served in the South African War of buboes which had been found to be due either to the thorns of the *Wachtenbitje* or to bites of ticks, to show that, even with symptoms suggestive of possible plague in times when the disease was endemic, it was necessory to eliminate sources of septic or other infection and to obtain confirmatory evidence by bacteriological examination by smears and inoculation of guinea-pigs.

He emphasized the need for a double-barrelled attack on the flea as well as the rat and showed how futile it was to attempt extermination of the latter, owing to the wonderful fecundity of rodents, but that *pari passu* with a wholesale intensive campaign at rat destruction and the plenteous use of kerosene as a pulicide it was necessary to concentrate on the rat-proofing of premises and the cutting off of the food and water supply of the rat by the regular removal and incineration of all food scraps and organic refuse.

PROFESSOR A. E. MILLS regarded the contributions they had heard on plague as very excellent and instructive. From Dr. Armstrong's paper it was evident that they in New South Wales were in a happier position than the people of Queensland in regard to the efficiency of the health control. The authorities in Queensland had had a good man in Dr. Burton Bradley, but they had not known how to keep him. He had admired the temperate manner in which Dr. Bradley had referred to the treatment accorded him. Although the curative effect of the serum had not been scientifically shown, the results obtained clinically were extremely encouraging. Turning to the question of diagnosis, Professor Mills referred to one patient whose condition had been diagnosed as pneumonia. The onset had been sudden, with a rigor. The patient looked desperately ill and was dull and apathetic. No physical signs were detected in the lungs. The heart was flabby and the pulse weak and rapid. There was a bubo which was not like an ordinary bubo; it was surrounded by a zone of œdema and swelling. Osler had said that suppuration of a plague bubo was a favourable sign. In the Sydney cases it certainly appeared as if this were true. Professor Mills referred to the extraordinary regularity with which the so-called serum sickness had appeared. He raised the question whether it should be regarded as an anaphylaxis phenomenon. It was difficult to explain on any other basis the prolonged latent period.

DR. C. W. REID referred to facts connected with the *Wyreema* incident. On September 19, at 9 a.m., the *Wyreema* arrived from Queensland at Sydney, having left Brisbane on September 17. At three o'clock in the afternoon five dead rats were found in No. 4 hold. Of these, one was found to be plague infected; the remainder were too putrid for examination. The gangways were immediately pulled up and the vessel was ordered into the stream for fumigation. The vessel was then emptied and was again fumigated. Twenty dead rats were found, of which eleven were plague infected. Dr. Reid pointed out that the *Wyreema* was the only ship with plague infection on board which had arrived from Queensland. He referred to the fumigation of ships known to be infected and the other methods applied, including rat guards. All gangways were drawn up at night-time. The rat population on board ships subject to fumigation diminished very rapidly and consequently the number of rats killed in any ship coming from Queensland had become very small. They had noted that rats frequently took refuge in the ship's boats. Dr. Reid asked whether pleural effusion had been observed in the plague-infected rats.

THE HONOURABLE J. B. NASH, M.L.C., as a member of the Board of Health, congratulated Dr. Armstrong on the important work he had carried out. He pointed out that the people who had to provide the money, had behaved very well in regard to this outbreak. The life or death of parliamentary parties was at stake. It spoke volumes that the responsible persons on all sides had agreed to give the President of the Board of Health the necessary authority to adopt all measures which might be required to destroy rats and to stem the epidemic. Relations between the Board of Health and Parliament had been of a cordial nature and everyone had been willing to do what was necessary.

DR. A. H. TEBBUTT, D.S.O., congratulated Dr. Burton Bradley on having prepared an anti-plague serum. He thought that it was advisable to place on record the exact qualities of the serum. It was important to know the number of different cultures employed in the immunization and to estimate the agglutination titre of the serum against these cultures. The best way of testing the efficacy of the serum was to use the serum prophylactically on guinea-pigs, rats or mice and to measure the amount of protection afforded. These experiments were not difficult to carry out. He hoped that Dr. Bradley would undertake them, so that the qualities of the serum might be placed on record and correlated with the clinical results. It was possible also that the serum might have some antitoxic qualities. In regard to the question of serum sickness, Dr. Tebbutt understood that at the Melbourne Hospital serum sickness was a somewhat distressing feature after administration of anti-pneumococcal serum. He suggested that this was rather a feature of antibacterial sera as compared with antitoxic sera.

DR. T. W. LIPSCOMB referred briefly to the girl patient at Parramatta. When he first saw her, she had not appeared to be very ill. Even a few hours before death she had not exhibited the typical facies of which Dr. Purdy had spoken, nor had she the appearance of a dying person. Dr. Lipscomb thanked Dr. Armstrong and the other speakers for their admirable contributions.

In his reply, DR. W. G. ARMSTRONG endorsed Dr. Nash's remarks in regard to the manner in which both sides of the Houses of Parliament made an effort to pass the necessary measures before the general election. They were thus enabled to bring into existence a code of plague regulations which had materially assisted the authorities in dealing with the outbreak. Dr. Armstrong referred more particularly to the formation of block committees and to the considerable amount of good work these committees had accomplished. He regarded it as very satisfactory that the total cost of the campaign had been kept to a very low figure without in any way sacrificing efficiency. He hoped that the work of the block committees would be permanent. They had acted throughout the business parts of the city. He would like to see the system extended to the business parts of the suburbs. He expressed the hope that the measures now being adopted would be successful in checking the present pandemic. He thanked the members for the kind things which had been said of the Department's work in connexion with the plague. The departmental staff fully deserved them. In that staff New South Wales possessed a body of officers of the very highest calibre. He thought that very few States had as fine a health departmental staff.

DR. EUSTACE FERGUSON, replying to a question by Dr. Reid concerning the occurrence of pleurisy in plague-infected rats, stated that other observers had claimed that pleural effusion was characteristic. At the Micro-Biological Laboratory they followed the routine described in his remarks and did not look for pleural effusion as a rule.

DR. BURTON BRADLEY agreed with Dr. Tebbutt that the laboratory tests for plague serum were not very satisfactory. He had experienced great difficulty in obtaining rats and consequently had not been able to test the protective power fully; and guinea-pigs, as was well known, were unreliable. In regard to the question of serum sickness, Dr. Bradley had deliberately avoided heating the serum and had not added any antiseptic. As it appeared to him that the presence of complement might have some therapeutic significance, he had relied on filtration to obtain sterility. He questioned whether the serum had strong antitoxic action.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

BRADLEY, GILBERT GEORGE, M.B., Ch.M., 1922 (Univ. Sydney), "Manfield," Johnson Street, Chatswood.

HOUEN, ANTONIO CHARLES, M.B., Ch.M., 1922 (Univ. Sydney), Cavendish Street, Stanmore.

MADDEN, JOHN PATRICK CAWLEY, M.B., Ch.M., 1922 (Univ. Sydney), Sydney Hospital.

PARK, ALEXANDER NORMAN, M.B., Ch.M., 1922 (Univ. Sydney), 3, Beaufort Street, Croydon.

READING, FANNY, M.B., B.S., 1922 (Univ. Melbourne), Belgrave Street, Kogarah.

SHAW, RICHARD GLINN VALLACK, L.R.C.P. (Lond.), 1921; M.R.C.S. (Eng.), 1921, Penshurst Street, Willoughby.

SHEPPARD, EDMUND MACARTHUR, M.B., Ch.M., 1920 (Univ. Sydney), c.o. University Club, Sydney.

SMALL, THOMAS HENRY, M.B., Ch.M., 1922 (Univ. Sydney), Sydney Hospital.

SUSMAN, ERIC LEO, M.B., Ch.M., 1921 (Univ. Sydney), "Rea Flats," Darlinghurst.

VERBRUGGHEN, ADRIEN HENRI PIERRE EUGENE, M.B., Ch.M., 1922 (Univ. Sydney), Guyra.

WINSTON, CHARLES EDWARD, M.B., Ch.M., 1922 (Univ. Sydney), Sydney Hospital.

NOTICES.

THE COUNCIL OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION has arranged the following provisional programme of the Branch. The Scientific Committee reserves to itself the right to modify the arrangements, but it is hoped that no changes will be necessary.

May 23, 1922.

At the Walter and Eliza Hall Institute, Melbourne Hospital, at 8.15 p.m., in conjunction with the Australian College of Dentistry Alumni Society.

DR. W. S. WILKINSON: "The Development of the Human Dental Mechanism." "The Significance of the Deciduous Teeth." "Orthodontia as an Aid to Pædiatrics."

DR. J. K. CLARK: "Oral Sepsis."

DR. J. M. LEWIS: "Cooperation Between the Medical and the Dental Professions."

June 7, 1922.

At the Walter and Eliza Hall Institute, Melbourne Hospital, at 8.15 p.m..

DR. W. J. PENFOLD will demonstrate the action of pneumococci on aromatic amino bodies.

DR. W. J. PENFOLD: "Australian Pneumococci."

DR. S. W. PATTERSON: (*a*) "Prognosis in Pneumonia"; (*b*) "Clinical Application of Serum Treatment of Pneumonia."

DR. R. L. FORSYTH will open the discussion on the serum treatment of pneumonia from clinical experience at the Children's Hospital. Papers will be illustrated by lantern slides.

Obituary.

ALFRED SUTTON.

THE news of the death of Alfred Sutton on April 15, 1922, which was recorded in our issue of April 22, 1922, occasioned much regret among the members of the profession in Queensland and also among many of those who served with the Australian Army Medical Corps in the late war.

Alfred Sutton was born at Egham, in Surrey, England, in the year 1863. Choosing medicine as his profession, he obtained the diploma of membership of the Royal College of Surgeons and was admitted as licentiate of the Society of Apothecaries in 1885. He started practice at Winchester and later moved to Seaforth, Liverpool. In 1888 he came to Queensland and settled at Beenleigh, where he practised for eleven years. From Beenleigh he went to Brisbane and practised there till his death.

Alfred Sutton's interest in military affairs began in 1900, when he was appointed Captain in the Army Medical Corps. Promotion came to him rapidly. He became Brevet Major in 1904 and attained the substantive rank of Major in 1907. After being Senior Medical Officer of the Northern Force and Commanding Officer of the Garrison Field Ambulance, he was promoted to the rank of Lieutenant-Colonel in 1909 and appointed to the position of Principal Medical Officer of the First Military District. From this time on till 1914 he was one of the few people who saw that war with Germany was inevitable and he did not tire in his efforts to train his unit and make its members efficient. When war broke out in 1914 he was one of the first to volunteer for active service and left Australia with the First Division as Officer Commanding the Third Field Ambulance. He held this command right through the Gallipoli campaign. After the evacuation Alfred Sutton assisted in the formation of the Second Division and became its first Assistant Director of Medical Services. He accompanied the Division to France in 1916 and was associated there with all its activities and hardships. He had a part in the fighting at Pozières and Mouquet Farm and went through the terrible winter on the Somme. He was with the Division at Bapaume and Bullecourt, at Menin Road and Paachendale and at the third Ypres struggle. He was mentioned in dispatches four times and was decorated after Bullecourt as Companion of the Most Honourable Order of the Bath. Subsequently he was awarded the decoration of Companion of the Most Distinguished Order of Saint Michael and Saint George. His health, however, gave way and he returned to Australia as an invalid early in 1918. He subsequently resumed his position of Principal Medical Officer of the First Military District and held it until the office was abolished in July, 1921.

Alfred Sutton was a successful practitioner of medicine and endeared himself to a wide circle of patients and friends. He had been Out-Patient Physician to the Brisbane General Hospital and also held the appointment of Honorary Physician to the Lady Bowen Hospital. He occupied the honourable position of President of the Queensland Branch of the British Medical Association in 1919. The Medical Defence Society was one of the organizations that claimed his attention. He was one of its foundation members, became its Secretary and Treasurer and sat for many years as a member of its Council. He was an ex-President of the United Services and the Johnsonian Clubs. At the time of his death he was Medical Officer to the Metropolitan Board of Water Supply and Sewerage. He is survived by his wife, a daughter and two sons. Both sons are in practice in Brisbane and they, together with his son-in-law, saw service with the Australian Imperial Force.

DR. G. P. DIXON, C.B.E., the President of the Queensland Branch of the British Medical Association, writes:

The members of the Australian Army Medical Corps, the medical profession and the people of Australia owe a great debt of gratitude to the late Colonel Alfred Sutton, C.B., C.M.G., V.D..

The fine work done during the war by the Australian Army Medical Service is a monument to his memory, of which, in his undemonstrative way, he was justly proud.

During his long term of office as Principal Medical Officer of the First Military District, his constant endeavour was to stimulate the medical men of Queensland to a realization of their duty to their country, in preparation for a war which he always declared was inevitable. Doctors and others occupied with diverse interests, were often inclined to scoff at his frequently-expressed opinion that we should be at war with Germany in our time and that it behoved every medical man to have a military training, so as to be prepared. He maintained that it was absolutely necessary for officers of the Army Medical Corps to be soldiers as well as doctors. He was right and many of us wished later that we had taken his advice.

During this period Colonel Sutton by precept and example did splendid work in organizing and training the Australian Army Medical Corps in Queensland. As a result of exhaustive study of the methods employed during the South African and Russo-Japanese Wars and his own observation during training operations in Australia, he published articles on the work of field ambulances which

aroused considerable attention at headquarters in Melbourne and at once stamped him as an authority on the organization and training of the Australian Army Medical Service. This peace-time work bore splendid fruit later on as we know.

When Britain declared for war, Colonel Sutton immediately volunteered for active service at very considerable personal inconvenience. Those of us who were fortunate enough to be chosen to go with him in the First Division, fully appreciated our luck in having the chance of serving under such a good officer. We all learned to love and respect our chief while training at Enoggera and in Egypt, for his high ideals, his absolute sense of justice, his sympathy with the "trier" and his scathing condemnation of the 'slacker" and above all for his unfailing courtesy and great kindliness to all in trouble.

As Commanding Officer of the Third Field Ambulance in Gallipoli he had his first opportunity of putting his theories to the test of actual warfare and well they stood the test. He also set officers and men an example of cheery optimism and scorn of danger, which had a most stimulating effect during those dark days.

His exceptional knowledge of administration soon gained him his well-earned promotion to the position of Acting Director of Medical Services to the Second Division, which he formed in Egypt, and successfully completely the arduous task of organizing and equipping the Army Medical Service.

It was while returning to Lemnos in the *Southland* that he went through the trying experience of being torpedoed, an incident which he often spoke of as a "most interesting experience"!

Colonel Sutton remained with the Second Division until the evacuation, endearing himself to all with whom he came in contact and gaining fresh laurels.

At Gallipoli he suffered severely from dysentery and epidemic jaundice and, through persisting in "carrying on" instead of seeking the rest and treatment he badly needed, he developed the heart mischief from which he suffered throughout his service in France and which ultimately caused his untimely death.

Others can tell of his distinguished career in the wider field of the campaign at the western front, where he gained his well-merited decorations.

An able man, a fine soldier, a true friend and a very courteous gentleman, Colonel Sutton gave all for King and Country and his sacrifice shall not be in vain—those who knew him will not forget, but will be the better for their knowledge.

ARCHIBALD LANG McLEAN.

DR. ARCHIBALD LANG MCLEAN, M.C., died at the Royal Prince Alfred Hospital, Sydney, on May 13, 1922, after a long illness. An account of his career will be published in an early issue.

Correspondence.

THE COST OF MEDICINES.

SIR: In making a comparison it is usually considered essential that the objects compared shall be of similar nature or at any rate possess some similar attributes.

Your correspondent, Mr. E. McC. S. Hill, writing on the above subject, implies that the action of a specialist in charging a higher fee for professional services than would a general practitioner is of exactly the same nature as that of a retail chemist endeavouring to obtain a higher profit for the sale of an article manufactured elsewhere than is usual among members of his profession.

Without again raising the subject of professional etiquette discussed by Mr. Hill, I would point out that the two actions are not of a similar nature and therefore not comparable. A specialist is entitled to an enhanced fee because his experience and knowledge of a particular subject is well in advance of that of the general practitioner, but it is not clear to me that qualilcation in pharmacy, however high, adds any value to the action of selling a proprietary preparation at a price above the rates for which it can be obtained by the public in the open market.

In conclusion, I trust that Dr. Jamieson will pardon me for entering upon the discussion of an aspect of a subject which he is better qualified to handle than I am. The presence, however, of what appears to be an illogical argument cannot but stir the reader of your correspondence columns to protest in some manner against any erroneous method of reasoning which may appear therein.

Yours, etc.,

GEORGE HAY.

"Oakleigh," Peak Hill, New South Wales,
April 24, 1922.

INFANTILE MORTALITY.

SIR: The following statement appears in the journal, *The World's Health*, published by the League of Red Cross Societies at Geneva in February, 1922:

"*Some Infant Mortality Statistics.*—The honour of having the lowest infant mortality rate in the world belongs to New Zealand, where it is now 40.6 per thousand. Dunedin in 1900 had an infant mortality rate of 80; to-day it is 30.8. This is ascribed to the mild climate, to favourable housing and economic conditions, to enlightened public health and welfare laws and to the excellent work of voluntary organizations in the provision of nurses, clinics and in educational propaganda for the conservation of child life."

For the year 1921 the Victorian rate was 72 and the Melbourne rate 74. In my judgement the New Zealand success is due to an excellent campaign of popular education directed at the subject from every conceivable point of view and to the fact that the New Zealand Government has largely placed the responsibility of managing the business in the hands of one medical man who is an enthusiast.

Yours, etc.,

JAMES W. BARRETT.

105, Collins Street, Melbourne,
May 5, 1922.

A THEORY OF CANCER.

SIR: Dr. Thomas Cherry's very interesting article on the theory of cancer is quite refreshing, considering how little research interest is taken in this disease in Australia. I would like, however, to point out a few facts that do not agree with Dr. Cherry's contentions.

Some years ago, when doing post-graduate study in America, I had the opportunity of following Dr. Wolf's experiments at Cornell University *re* sarcoma and two of his findings were:

(1) If a sarcoma were subjected to high pressure in an improved Berkefeld filter and the filtrate injected into a guinea-pig (I saw seven inoculated), no growth follows.

(2) If in the filtrate one allowed a minute amount of cell tissue, one got a growth, proving that the filtrate or really protoplasm itself was not sufficient to produce growth.

Following this up, it is noticeable that cancer starts as a simple tissue, but rapidly changes its character and becomes a new formation, *i.e.*, a rebuilt cell, with much activity; it is this complete change of cell and character that marks the beginning of trouble. If this were due to an irritating toxin like "proliferin" we would probably get increased activity, but the cell would remain unchanged and a new growth would follow a filtrate injection. This we know by experiment does not happen. An accumulation of cells, such as a kerostosis, which, like *leucoplakia oris* or *verruca seborrhœica*, is often a forerunner of cancer, requires a local bio-chemical reaction to cause it to assume a cancerous character and until such reaction, it remains innocent and cannot be transmitted by injection into fresh tissue.

Further, if we treat an epithelial accumulation with a soft X-ray tube, one will find immediate cell activity in the majority of cases and if continued will gradually show signs of malignancy. If a high[1] tube is used with sufficient

[1] Dr. McLeod probably means a hard tube.

power, one gets a cessation of cell activity and a "destruction" or rather change of cell and cell content. Again, if perfectly healthy tissue is treated (especially with a soft tube) continuously, one will get an X-ray ulcer which rapidly developes all the characteristics of malignancy, all of which, in my opinion, points to an electro-bio-chemical theory of tissue change.

After wandering on the surface of this interesting study for some time, I suggest that cancer is caused by a breakdown and reformation of the electro-bio-chemical ties due to local disorganized ionization. This disorganization is due to some form of destructive vibration, such as various forms of injury, light or electro-chemical stimuli. By electrical stimuli I mean inter-cellular ionization, which probably acts in a similar manner to the bombardment emanating from the cathode of an X-ray tube.

Yours, etc.,

A. REGINALD MCLEOD.

143, Macquarie Street, Sydney,
April 26, 1922.

Books Received.

CANCER AND ITS NON-SURGICAL TREATMENT, by L. Duncan Bulkley, A.M., M.D.; 1921. New York: William Wood & Company; Royal 8vo., pp. 457. Price: $6.00 net.

ANNUAL REPORT OF THE SURGEON-GENERAL OF THE PUBLIC HEALTH SERVICE OF THE UNITED STATES FOR THE FISCAL YEAR 1921. Washington: Government Printing Office; Demy 8vo., pp. 429, with maps, diagrams and tables.

MEDICAL AND SURGICAL REPORTS OF THE EPISCOPAL HOSPITAL, PHILADELPHIA: Volumes IV. and V.; 1916 and 1917-1920. Philadelphia: Wm. J. Dornan; Royal 8vo., pp. 326 + 506, with 109 + 162 figures.

TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA: Third Series, Volume Forty-Second; 1920. Philadelphia: Printed for the College; Royal 8vo., pp. 519.

Medical Appointments.

DR. C. O. G. DONOVAN, D.S.O. (B.M.A.), has been appointed Government Medical Officer at Culcairn, New South Wales.

* * * * *

THE undermentioned have been authorized by the Board of Health, New South Wales, as Inspectors under Section 50 of the *Cattle Slaughtering and Diseased Animals and Meat Act, 1902*: DR. T. O. F. ALSOP (B.M.A.) at Binalong, DR. H. V. GILLIES (B.M.A.) at Coonamble; DR. R. S. R. MITCHELL (B.M.A.) at Gulargambone, DR. M. F. O'REILLY (B.M.A.) at Bega, DR. E. H. WHITE (B.M.A.) at Candelo, DR. W. F. S. YEATES (B.M.A.) at Ballina.

WE are informed that DR. BURTON BRADLEY and DR. HAROLD NORRIE are no longer associated with the Biological Institute of Australasia and that products sold by that firm are no longer subjected to physiological control by them.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii..

PEDFORD PARK SANATORIUM FOR CONSUMPTIVE SOLDIERS, SOUTH AUSTRALIA: Medical Officer.

ISISFORD DISTRICT HOSPITAL (CENTRAL QUEENSLAND): Medical Officer.

PARKSIDE MENTAL HOSPITAL, SOUTH AUSTRALIA: Junior Medical Officer.

PUBLIC SERVICE COMMISSIONER'S DEPARTMENT, QUEENSLAND: Medical Officer for Venereal Clinics and Venereal Isolation Hospital, South Brisbane.

UNIVERSITY OF MELBOURNE: Stewart Lecturer and Demonstrator in Pathology.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Societies Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Manchester Unity Independent Order of Oddfellows Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Hampden District Hospital, Kuridala, North Queensland Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 3, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

MAY 23.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
MAY 24.—Victorian Branch, B.M.A.: Council.
MAY 25.—South Australian Branch, B.M.A.: Branch.
MAY 25.—Brisbane Hospital for Sick Children: Clinical Meeting.
MAY 26.—New South Wales Branch, B.M.A.: Branch.
MAY 26.—Queensland Branch, B.M.A.: Council.
JUNE 2.—Queensland Branch, B.M.A.: Branch.
JUNE 7.—Victorian Branch, B.M.A.: Branch.
JUNE 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 9.—Queensland Branch, B.M.A.: Council.
JUNE 9.—South Australian Branch, B.M.A.: Council.
JUNE 10.—Eastern District Medical Association, New South Wales.
JUNE 13.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 14.—Western Australian Branch, B.M.A.: Council.
JUNE 14.—Melbourne Pædiatric Society.
JUNE 15.—Victorian Branch, B.M.A.: Council.
JUNE 16.—Eastern Suburbs Medical Association, New South Wales.

Editorial Notices.

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